The Journal of HEREDITY

MIDGET-EYED HOMER
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Mouse Genetics News

A "Milk RH Factor" In Horses

New Cowpeas for the Tropics

No Type III Cowlicks Among "Mongolians"

The True Story of the Ohio "Octuplets"

Pedigrees of "Smell Blindness"

VOLUME 39 OCTOBER, 1948 NUMBER 10

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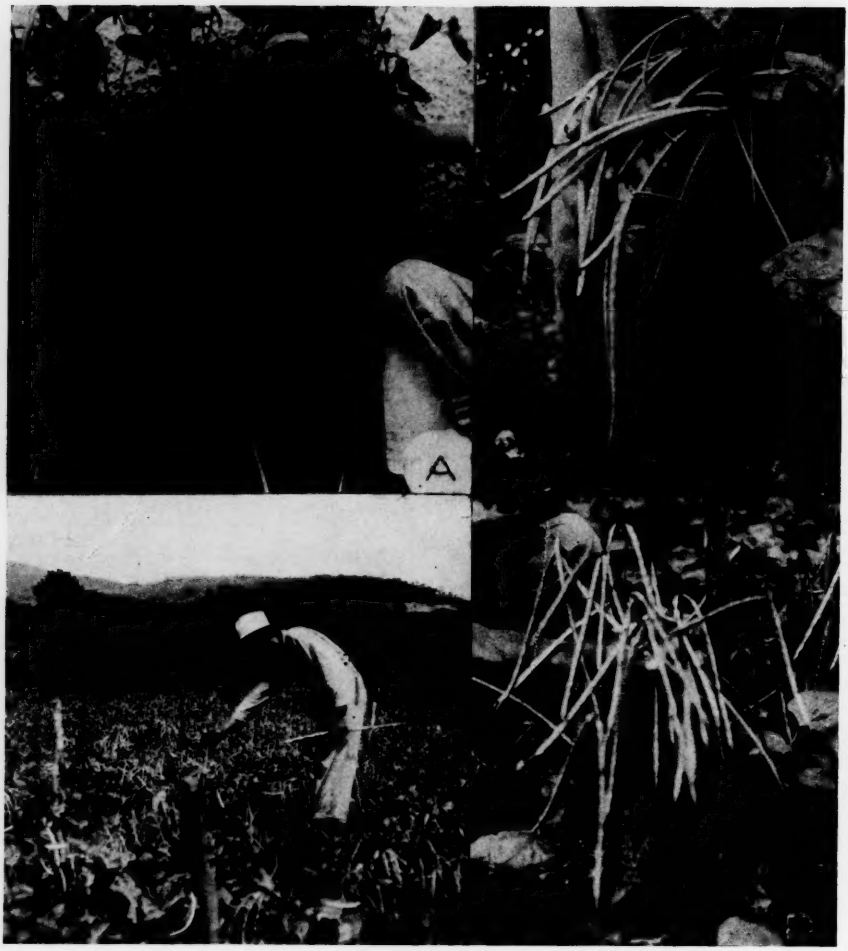

COWPEA HYBRID SELECTIONS

Figure 1

A—The "Yardlong bean" from which mildew resistance was obtained. *B*—First generation hybrid of New Era cowpea with yardlong. Note size of pods. *C*—Making individual selections in a planting of the new Institute variety "Chinito." Note heavy production and foliage-free pods. *D*—Individual field selection from the cross of New Era with yardlong crossed again with the new Institute variety Azul Grande.

NEW COWPEAS RESISTANT TO MILDEW

JOSEPH L. FENNELL

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THE importance of vegetable protein foods and more specifically of the bean (*Phaseolus vulgarus*) and its relatives in the diet of tropical countries is well understood. It is an important mainstay of life throughout many of the warmer regions of the Americas. But due to various climatic and cultural difficulties the bean has not always been fully successful in certain locations or in certain seasons. This, especially, has been the case wherever the climate has tended toward extremes in drought or humidity.

As a consequence of lack of adaptation of the common bean to these conditions tropical agriculture has been faced with the responsibility of finding a complimentary and auxiliary substitute: a plant having equal edible and nutritive value but possessing the ability to grow and to produce in regions and under conditions not favorable for bean culture. The difference between health and hunger for large populations has hinged on the successful solution of this problem.

Of the many leguminous crops tested in our experiments in Costa Rica, the cowpea (*Vigna sinensis*) has given greatest promise of an ability to fulfill this need. This was true, however, only in the broadest sense, since several obstacles have also stood in the way of using this crop. Notwithstanding this, from the plant breeders' viewpoint the genus *Vigna* seemed to encompass more needed and usable breeding values for tropical production than were available within the more restricted species limits of the true bean and its inter-breeding relatives. The difficulty of hybridizing the bean with most other species has greatly hindered its improvement.

In our cowpea project at the Institute, twenty-eight commercial varieties and many experimental and primitive sorts have been tested. Two varieties have shown the most promise in hundreds of plantings over a period of more than four years. These are the New Era for consistent high seed yield and superior tolerance to mosaic infection, and the Sugar Crowder for superior edible qualities with at least fair production. A few other varieties, notably the Conch, Potomac and Browning Crowder, have fair production and have indicated merit when employed as breeding parents.

New Era is one of the most productive varieties grown in many sections of the southern United States.[3] Moreover, the variety has been reported as the most satisfactory cowpea of many kinds tested in the Philippine Islands. Despite its superior traits, New Era has several faults, notably the small seeds and relatively poor table quality. Nevertheless its use as a breeding parent seemed well justified, a conclusion that has been amply supported by actual breeding results.

Breeding and Improvement

In this project many controlled crosses have been made with three principal objectives in view: 1) better disease resistance; 2) higher year-round seed production in hot humid climates; and 3) better quality and appearance for table use.

From crosses between New Era and Sugar Crowder two new varieties have been produced that show promise. One of these which we have named Garbancito (No. 0151) produces a large seed of cream color which very closely resembles the Sugar Crowder parent. The plants have consistently been much more vigorous, healthy and productive, than has the parent variety. Another selection which we have named Azul Grande (No. 0150) has the large seeds of Garbancito but with the gray-blue color of New Era.

In preliminary trials, very encouraging and even superior yields have been given by these new varieties. In some cases yields have surpassed those of New Era and under the humid weather conditions of the autumn months at Turrialba, Costa Rica, they have consistently exceeded bean yields by many times. The tests are still in process, but all indications are that these new sorts may prove more satisfactory than any table variety of cowpea yet tested.

These selections from New Era crosses have shown more tolerance to mosaic diseases than has been noted in the majority of cowpea varieties in our tests. Flavor and general table quality of these new kinds is good. Seed is now available for trial distribution in sample quantities.

From crosses between Conch and Potomac several interesting selections have been made, though none of these has been outstanding. One new variety (Canela No. 0128) gives good production of seeds of light cinnamon color. It makes a rank and vigorous growth and may prove of value for hay or cover crop purposes.

In many parts of the Americas, powdery mildew (*Erysiphe polygoni*) has been most destructive in cowpea plantings under certain weather conditions. Most of the cowpea varieties (*V. sinensis*) tested in our trials have shown about equal susceptibility to this disease. At Turrialba, the relatively cool and dry months from November to April are the most favorable season for cowpea production. But even then plantings are frequently rendered worthless by mildew attack. It has been reported by Brown[1] that when grown on soil with a pH from 4.8 to 6.4, cowpeas are virtually free from mildew. At Turrialba we find that all susceptible varieties have been badly attacked on soil of this pH range. This and other widely varying results lead to a belief that distinct strains of *Erysiphe* may exist.

Indications are that cowpea mildew is widespread and that it has been one of the most serious obstacles confronting production of this crop in many sections of tropical America.

Mildew-Resistant Hybrid

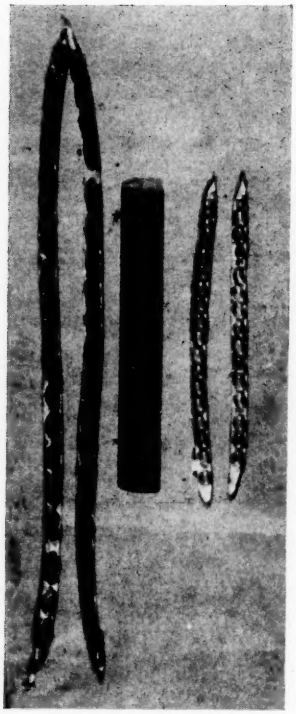
The problem of mildew control was largely solved through the use of another related species (*V. sesquipedalis*), popularly called "yardlong," "china bean" or "asparagus bean." The mildew resistance of this species has been reported[2] and selections gave indication of possessing near immunity to *Erysiphe*.

Yardlong pods are much longer than are those of cowpea, typically measuring from one and one-half to three feet in length. The seeds are proportionately longer than those of the cowpea, showing much the shape of a kidney bean. Plants of the yardlong are high climbing and viney in growth and produce roots and stems considerably stronger than those of cowpea varieties. For this reason seed production per plant has been relatively large.

When the yardlong was crossed with cowpea varieties, segregates obtained in the second and third generations combined many of the best characteristics of both species. A few lines have shown the following complementary values: medium large pods well filled with good sized bean-shaped seeds; heavy production with pods held above the foliage; bush or near bush habit; fair tolerance to mosaic; and what is even more important, high resistance to powdery mildew. In the third and later generations some lines have shown virtually uniform resistance to the mildew fungus.

Inheritance Observations

Notes on the inheritance of a number of characters compiled during our cowpea breeding project may be of interest to others working toward improvement of the crop. When the variety Conch (white flowers and white seeds) was crossed with Potomac (nearly white or slightly purple-tint flowers and white seeds with brown eye) all F_1 progeny produced dark purple flowers and seeds of solid brown color. The F_2 population of 63 mature plants segre-



SPACING DIFFERENCES

Figure 2

Mature pods with semi-Crowder seed spacing of New Era (at right), compared to pods with normally spaced seeds of the F_1 hybrid between New Era and yardlong.

gated with respect to flower and seed color in a simple Mendelian ratio as follows: tinted flowers and brown eyed seeds, 15 plants; purple flowers and solid brown seeds, 34 plants; white flowers and white seeds, 14 plants. This association between seed color and flower color has been supported by later and larger populations.

When the purple-flowered and brown-seeded segregates noted above were crossed with the black-seeded yardlong, a low percentage of blackeye, browneye, white-, yellow- and pink-seeded forms appeared in the F_2 and later generations. Black seeds have predominated in a ratio of higher than 16 black to 1 other color in F_2, indicative of multiple factor dominant inheritance for black seed coat.

In crosses between New Era (blue seed) and Sugar Crowder (yellow seed), F_1 plants showed the dominant blue seed color of New Era and normal spaced seeds. F_2 segregated 1392 blue: 88 yellow, approximately a 15:1 ratio. Certain individual plant selections made in F_2 produced in F_3 1125 blue:360 yellow, approximately a simple 3:1 ratio. Other blue-seeded selections of the same cross produced in F_3 the 15:1 ratio. This indicates that two dominant genes carry the blue color of New Era. The crowded (Crowder type) seed spacing has behaved as a complex recessive, giving two or more degrees of this Crowder character in the F_2 progeny. The frequency of recovery of the full Crowder phenotype has not been determined.

In crosses between the yardlong bean and the New Era cowpea, a population of 127 F_1 fruiting progeny derived from parents homozygous for the contrasting characters noted, the following characters were observed and their dominant relations in F_1 and distribution in F_2 is given:

Character	*Dominant in F_1*	*F_2*
Climbing vs. bush	Climbing	Multiple factor
Long black seeds vs. short blue seeds	Long black seeds	Multiple factor
Mildew susceptibility vs. mildew resistance	Mildew susceptibility	Multiple factor
Long pods* vs. short pods	Long pods	Multiple factor
Medium (not crowded) seed spacing vs. wide spacing of yardlong parent	Medium spacing	Multiple factor

*The extremely long (30 in.) pods of the yardlong parent represent expression of a combination of factors: long pod determined by size, shape and number of seeds, inherited as a dominant; and wide seed spacing, inherited as a recessive.

Resistance to mildew has segregated as a multiple factor recessive. The varying degrees of mildew susceptibility evident in the F_2 and F_3 populations has led to the belief that two or possibly three genes are involved. In an F_2 population of 824 mature plants, 778 showed strong to moderate mildew attack and only 46 were relatively free of the fungus. Even these resistant segregates

were not always completely free of mildew, for close examination revealed slight traces of *Erysiphe* along the older petioles of some plants. Resistant individual plant selections made in the F_2 generation have given F_3 populations with comparatively uniform resistance to mildew. These data support the hypothesis that two factors may be responsible for major susceptibility; one or more other factors influence relatively minor susceptibility.

It may be of interest that this situation is contrary to that previously reported[2] for crosses of the yardlong with cowpea varieties. In this earlier report it was stated that resistance was inherited as a simple Mendelian dominant. These conflicting results further suggest the possibility of distinct genetic strains of the pathogen.

A New Race of Cowpea Varieties

Many lines derived from these crosses between the yardlong and various varieties of cowpea are now being grown. A few of the best selections have shown almost homozygous resistance to mildew, nearly uniform bush habit of growth and heavy production of closely spaced large seeds. Colors have been mostly black or blue.

From a cross between yardlong and a derivative of a Conch × Potomac cross, lines were selected which give large kidney-shaped beans in white, yellow, brown, red, white with yellow eye, and white with black eye. All black eye, brown eye, or pure white selections or varieties, irrespective of genetic background, that were tested or developed in our project, have given considerably poorer yields, especially in humid weather, than have those with other seed colors. This correlation has been maintained even in selections derived from the progeny populations that have given superior strains with other seed colors.

In the third generation of yardlong × cowpea, selections highly resistant to mildew and superior in other agronomic characters were crossed with our new cowpea variety Azul Grande, previously mentioned. From this backcross to the cowpea some novel and very promising selections have been made. One new variety, (Chinito, No. 0190) is extremely productive. Its large pods filled with light blue semi-crowder type seeds are held on strong peduncles well above the leaves. This is a characteristic typical of most yardlong hybrids and one of considerable importance in humid climates. This variety is highly resistant to mildew and is moderately free of mosaic. Edible quality is good.

Another selection (No. 0198) from this cross has cream-colored seeds, in general similar to the sugar crowder grandparent but resistant to mildew. Selection No. 0189 from this same cross has been highly productive of medium large, black, bean-like seeds. The black-seeded selections may prove of especial value in the regions where black beans (*Phaseolus*) are preferred.

The principal asset of these yardlong × cowpea crosses is their resistance to mildew. With freedom from this serious disease the geographical range of cowpea production may be extended advantageously.

The Cowpea in Tropical Agriculture

The question frequently is raised as to the relative value of cowpeas, soybeans and the common bean for culture in equatorial regions. Because of its high protein content and general nutritive value, the soybean has frequently been championed. Despite its merits and much favorable propaganda the soybean as a food crop has not been favorably accepted in the warmer parts of this hemisphere. Probable reasons for this poor acceptance, and factors that have led us to make an important project of the cowpea follow:

1. In tropical America beans and kindred foods are usually utilized in the dry condition. When dried, the common soybean is tough, hard and requires extremely long cooking to be made palatable. Fuel must be utilized to best advantage since often it is costly or diffi-

cult to obtain. The more easily cooked softer-seeded soybean varieties have given consistently poor yields in our tropical tests.

In important contrast with this serious soybean cooking problem, dry cowpeas cook readily and require less time than the ordinary bean to become ready for eating.

2. The cowpea is less sensitive than the soybean to adverse tropical cultural conditions, such as deficient soil fertility, extremes of rainfall and drought, and apparently to short daylight periods. Soybean harvesting problems and the probability of loss from shattering or moulding in very dry or wet weather seem also to be greater.

3. To be of value as a soil builder, the soybean or its seedbed must be inoculated with proper bacteria, which in our tests were not locally available. This inoculum has not persisted in the soil.

In comparison, bacteria for cowpeas are widely available, due to the great assortment of native and related wild plants. In our trials in Costa Rica a previous planting of cowpeas showed a more favorable influence on the following crop than did crop sequences of soybeans or common beans, even after inoculation.

4. In its crude state the cowpea can be utilized directly in more ways than the soybean as food for humans, poultry and livestock.

Results of our experiments in Costa Rica and consideration of many modifying circumstances lead to the conviction that the cowpea has greater value as a direct-consumption food crop for tropical America than has any bean relative known at present. It is always possible of course that, with better breeding material or with improved cooking methods, this relative standing of the two plants could be materially changed.

Summary

The need for an auxiliary or complementary substitute for the bean in tropical regions seems best realized in the cowpea. The varieties New Era, Sugar Crowder and the "Yardlong" (*Vigna sesquipedalis*) have given good results as breeding parents.

Two promising new crowder varieties, "Garbancito" and "Azul Grande", have been produced.

Resistance to mildew has been obtained through crosses with yardlong. Crosses of these yardlong hybrids with the new cowpea variety Azul Grande gave several highly promising selections from which new varieties are being perfected.

Notes on inheritance, and comparison of cowpeas with soybeans for tropical needs and culture are offered.

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DIABETES MELLITUS IN IDENTICAL NEGRO TWINS

And the Association of Taste Blindness and Diabetes

THE factor of heredity in the incidence of diabetes mellitus, recognized as long ago as 1689,[2] is of special interest to the modern student. It engages the attention of the latest writers as may be seen in a recent report by the United States Public Health Service[6] on its first community study of diabetes in the United States. In 1937 personnel of that Service made a case-finding survey of a town of 4983 inhabitants in Massachusetts. Those assigned to the task saw and examined 70 per cent of the entire population. The examination included urine analysis, blood sugar determination and inquiry into family history. On the point of history the report is as follows: "A family history of dia-

betes was found in 38.6 per cent of the cases of diabetes and in only 18.2 per cent of the non-diabetic cases."

In 1932 Priscilla White[5] reported diabetes in eighteen pairs of twins, fifteen of which pairs were identical, or monozygotic. In ten of the pairs of identical twins the onset was almost simultaneous. In five pairs there was an interval of from five to thirty years.

The writer has had an opportunity recently to see a pair of identical twins with diabetes, of special interest because the patients are negroes.[3, 4]

Mrs. F. D. H. and Mrs. A. H. J. are negro (mulatto) women about sixty years old, born in Mississippi. Since 1915 the two have lived, most of the time, as far apart as Palo Alto, California, and Brooklyn, New York, but the comfortable economic status of both families has permitted several lengthy visits between them. Both patients have had adequate medical attention including insulin therapy. The diagnosis of diabetes in the case of Mrs. H. is on file in the Palo Alto Clinic dated April 12, 1937. A history of "constant thirst" makes it probable that the disease began in her case as far back as 1931.

In 1937 Mrs. A. H. J. came to visit her sister in Palo Alto. Just before leaving home she had a check-up by Dr. C. C. L. of Brooklyn, who, (quoting Mrs. J.) found no sign of diabetes. Of her visit in Palto Alto Mrs. J. says: "my sister had a fig tree and I constantly ate a great amount and also more fruit than ever." On her return to Brooklyn in the same year Dr. C. C. L. found she had diabetes. It appears. therefore, that a period of perhaps six years may have intervened in the recognition of the disease in these two persons of identical susceptibility.

The writer saw the twins on two occasions at the home of Mrs. H. in Palo Alto. Their likeness was astonishing. Mrs. H. was in bed at the time with severe arterial hypertension and measurements of any kind were out of the question—with one exception to be mentioned later. In spite of this circumstance the requirements of Newman's Similarity Method[1] for the diagnosis of monozygosity were well met, except for fingerprinting, by information furnished by the staff of the Palo Alto Clinic and others.

The one comparison of a laboratory nature, spoken of above, was that made by the writer to determine taste reaction to phenylthiocarbamide (PTC). Both patients, tested in separate rooms, immediately recognized a bitter taste.

Recently Terry and Segall[4] have reported an association of tasteblindness with diabetes in a population of 680 persons, white and negro. Among the 211 white diabetics 41.3 per cent were tasteblind; among the 301 non-diabetics only 25.9 per cent. The difference, 15.3, is highly significant. Among the 168 negroes seen only 11 diabetics were found, all tasters of PTC. Among the 157 non-diabetic negroes only 20.4 per cent were non-tasters.

The present communication brings the total of Negro diabetics in these studies to 13 by adding 2 newly discovered cases of especial interest because the patients are identical twins.

The complete absence of taste blindness in a group of 13 Negro diabetics suggests a racial difference with respect to diabetic susceptibility and PTC sensitivity but the numbers are far too small to warrant statistical consideration. However they represent a tiny addition to the evidence that the relationship between PTC sensitivity and diabetes is a function of the total genotype.

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FRONTAL HAIR DIRECTION IN MENTALLY DEFICIENT INDIVIDUALS

With Special Reference to Mongolism

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IN A previous paper in this Journal[2] it is shown that the different types of frontal hair direction in man normally appear to be dependent on autosomal multiple allelomorphic genes, probably acting on skin growth at a particular stage of fetal life. The different types of frontal hair slopes are probably the result of an interaction between two growth-directing centers, one near the root of the nose, the other in the area of the crown. If the growth impulse tending from the first region has its maximum activity, the hair slope will form the strongest dominant type III; if the growth directed from this region is minimal, the result will be the weakest recessive type I. Intermediate strength of the growth activity from this area forms the more variable type II.

Investigations of monozygotic twin pairs show that, though they always show concordance in major type of frontal hair direction, there may occur small variations in the hair pattern, probably caused by small differences in growth rhythm, which in turn may be induced by differences in the environments of the two fetuses.

It would therefore be possible that alterations in the head hair pattern could be found in connection with abnormal growth of the skull. This paper deals with some of the results obtained from an investigation of 336 mentally deficient individuals, including 66 unquestionably mongoloid.

The Different Groups of Patients Examined

The patients investigated were nearly all born in the State of Connecticut, U. S. A., and represented different kinds of mental deficiency. As most of the diagnosed specific cases, like hydrocephaly, microcephaly and cretinism, numbered too few for a special sorting, the separation according to diagnosis is made here only between mongoloid and non-mongoloid patients. All the diagnosed mongoloids at the institution were examined. Of the non-mongoloids this investigation includes 270 out of about 1,300; only about 10 per cent of these showing head deformities.

As it has been supposed that mongolism occurs with different frequencies in different ethnic groups, I have noted the native country of the grandparents of the patients examined (Table I). Both in this table as well as in Table II the Eastern European group also includes Poland, Czechoslovakia, Hungary and Greece. The geographical distribution of the ancestors of the mongoloid patients seems to be in good accord with the relative frequencies of these geographical groups in the ancestry of the whole population in this State with the exception of the Eastern European group which shows a considerably higher percentage than should be expected.

Only one of the mongoloid patients had colored near ancestors (½ American Indian, ¼ American Negro, ¼ white). In agreement with the experience of Schlapp,[4] this material shows that mongolism is at least as common in the Jewish population (7 patients of 66 were of Hebrew religion, most of their grandparents were born in Eastern

*This investigation was made possible through the cooperation of Superintendent Neil A. Dayton, M.D., and his staff, of The Mansfield State Training School and Hospital, Connecticut.

I also acknowledge with pleasure my indebtedness to Dr. W. Landauer, Head of The Department of Genetics, The University of Connecticut, and to Dr. E. Zwilling at the same institute for their helpful hospitality during my stay as a guest at Atwater Laboratory, Storrs.

MERGING GLABELLAR STREAMS

Figure 3

Normal one-egg twins of type III. These young ladies show the typical frontal hair pattern with the glabellar stream spreading fanwise and reaching the scalp border on both sides of the forehead. Type III, which is found in more than a quarter of non-mongoloid populations, is not observed in the mongoloids.

Europe) as it is in the other whites.

Further, as it is to be expected that the frequencies of the different types of frontal hair direction will vary according to the different ethnic groups, I have tried to place some of the non-mongoloid individuals in ethnic-geographical groups (Table II) according to the birthplace of their grandparents and their individual skin color (white and Negro).

Frontal Hair Directions in the Different Groups

Table II shows the distribution of the major types of frontal hair direction within different geographical and ethnic groups of non-mongoloids.

In the first group are those whites whose grandparents were all born in North America (before the intense immigration from Southern and Eastern Europe) and/or in Northwestern and Central Europe. This group shows frequencies (42.1 per cent of type I, 31.6 per cent of type II and 26.3 per cent of type III) similar to those found in normal Norwegians (42.5 per cent of type I, 28 per cent of type II and 28 per cent of type III with 1.5 per cent undetermined).

In the second group the individuals of pure Italian extraction showed a considerably higher frequency of type III (36.4 per cent) and proportionally lower frequency of type II; while the third group of entirely Eastern-Euro-

TABLE I.—The nearest ancestry of the 66 mongoloids investigated.

Ethnic group of grandparents	Number	Per cent
White, born in:		
1. North America	94	35.6
2. North-Western and Central Europe	64	24.2
3. Eastern Europe	62	23.5
4. Southern Europe	12	4.5
5. Birthplace unknown	29	11.0
American Indian	2	0.8
American Negro	1	0.4
Total	264	100.0

TABLE II.—Frequencies of the major types (I, II, III) of frontal hair directions in non-mongoloid mentally deficient individuals from different ethnic and geographical groups.

Group	Type of frontal hair direction						Total
	I		II		III		
	n	%	n	%	n	%	
1. White, with all their grandparents born in:							
a. North America, North-Western and Central Europe	48	42.1	36	31.6	30	26.3	114
b. Southern Europe (Italy)	14	42.4	7	21.2	12	36.4	33
c. Eastern Europe	17	44.7	15	39.5	6	15.8	38
2. Colored:							
a. American Negro	10	32.3	3	9.7	18	58.0	31
b. American mulatto	9	75.0	1	8.3	2	16.7	12

NEARLY IDENTICAL COWLICKS

Figure 4

If the glabellar stream surpasses the border of the scalp, the result may be "cow-licks" like in these normal one-egg twin boys of type III.

pean descent showed a very low frequency of type III (15.8 per cent) and a proportionally higher percentage of type II.

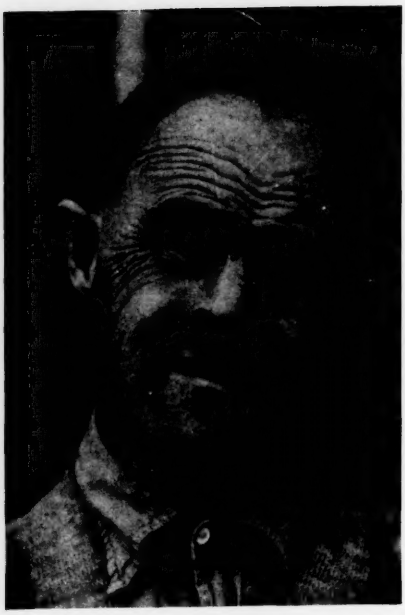
Although admittedly the present material is not sufficient for a final determination of the relative hair pattern frequencies in populations of the respective parts of Europe, it still suggests that type III may be less frequent in eastern Europe and more frequent to the south.

The non-mongoloid colored patients examined in the same institution were sorted into two groups. In the first are those whose ancestry is unknown, or of whom it is stated that all their near ancestors were American Negro ("American Negro" in Table II). The other group contains those who are stated to have near ancestors also from white and/or American Indian stock ("American mulatto" in Table II). It is interesting that the first of these two groups, which contains 31 individuals, shows the largest frequency of type III found in any of the ethnic groups, namely, 58 per cent, while the smaller group of individuals with probably fewer of the "African genes" shows only 16.7 per cent of type III. So far we do not know the distribution of the different types of frontal hair direction in normal American Negroes, but this material suggests that type III may be frequent in this population.

TABLE III.—Frequencies of the major types (I, II, III) of frontal hair directions in mentally deficient individuals: 227 white non-mongoloid and 66 mongoloid.

Group	Type of frontal hair direction						
	I		II		III		Total
	n	%	n	%	n	%	
1. Non-mongoloid white	98	43.2	68	30.0	61	26.8	227
2. Mongoloid	42	63.6	24	36.4	0	0	66

With these observations as a background, we will now turn to Table III, which shows distribution of the three major frontal hair pattern types in all the examined non-mongoloid white and all mongoloid patients. Of the total 227 non-mongoloid white, 43.2 per cent showed type I; 30 per cent, type II; and 26.8 per cent, type III. If we calculate from this last percentage as typical, we should expect about 17 individ-



MONGOLIAN WITH TYPE I HAIR STREAM

Figure 5

48-year-old male with the physiognomical traits attributed to mongolism: oblique eye position, epicanthus, cataract on both eyes, slightly deformed ears with partly attached earlobes, protruding under-lip and tongue, flat occiput, depressed nose root, stub-nose, and straight, scanty hair. The strongly corrugated skin is a sequence of the typical spacious skin condition in youth. This man also has the short fingers, the hooked little fingers, the simian crease in both palms and the extremely short stature (149.8 cms.) so common in mongoloids. His I.Q. is 24. Like 63.6 per cent of the mongoloid patients he shows type I of frontal hair direction.

uals of type III among the 66 mongoloids. If we consider the different frequencies in the different ancestral groups (Tables I and II), we should expect 16 of the mongoloid patients to show type III. It is then surprising to learn that *type III is quite absent in the mongoloid group.*

Discussion

The difficulty in explaining the absence of type III frontal hair slope in the mongoloids in relation to any of the existing hypotheses for the etiology of mongolism lies in the fact that we do not know if the gene, normally producing type III, really is absent in all these mongoloids, or if its manifestation is prevented by other factors. A family investigation of the mongoloids would be desirable, although it may not be easy to determine if one of the parents of a mongoloid child is homozygous for the gene producing type III. Nor do we, unfortunately, yet know any linkage between the gene normally producing type III and other physical or physiological genetic factors. If such a linkage should be found, it may be possible to decide if the gene D^{III} really is missing in the mongoloids. The possibility that further material may reveal mongoloids showing type III is of course also to be considered.

Whatever the case may be, it is highly probable that the absence of type III frontal hair direction in the mongoloid patients is associated with disturbance in growth rhythm during an early fetal stage. In this connection it is interesting that Ingalls,[1] from his studies of the pathogenesis of mongolism, concludes that "the time of origin may be narrowed down to a period lying between pre-embryonic and early embryonic life (probably before the third month) of the affected person." Nehse[3] found that the first hair primordia in man appear on the forehead towards the end of the second embryonal month and the penetrance of the frontal hairs through the epidermis occurs at the end of the third month; whereas the hair primordia on the scalp become apparent at the end of the third month, and the hairs here penetrate the epidermis in the middle of the 5th month.

Early in the eighth week the cartilaginous cranial floor of the skull with nasal capsules has already appeared and during the next week the endochondral ossification centers of the nasal and frontal bones appear. The depressed glabellum and nasal bones found in mongoloid patients are supposed to be caused by a retarded development of the con-

tiguous ethmoid part of the cranium.[1] As the ethmoid parts of the chondrocranium normally are found already in the early part of the eighth week, there may be a possible causality between a retardation of growth in this part of the cranium and the formation of the hair slope in the glabellar region.

The other growth-directing center, determining the crown stream, seems not to be much affected in the mongoloids. It is of interest that the 65 mongoloids (one was quite bald) showed 9.2 per cent of double occipital whorls, while 14.3 per cent of the 223 non-mongoloids showed this aberration. In normal Norwegians I have found 8.5 per cent with double crowns.

Therefore, if the skin growth normally forming the glabellar hair stream is affected by the retardation of growth in this part of the head, while there may be no such retardation in the growth activity directed from the crown, the crown stream will prevail and give the type I or type II of frontal hair slope.

Summary

With regard to the different types of frontal hair slope, 336 mentally deficient individuals, including 66 mongoloids, were examined.

The 227 non-mongoloid white patients showed frequencies of different types similar to those found in normal Norwegians. Sorting according to their ancestry showed higher frequency of type III in individuals of Southern European origin and lower frequency of type III in those of Eastern European extraction; while those whose ancestors were born in Northwestern and Central Europe showed distribution of frontal hair pattern similar to that of Norwegians.

The American Negroes investigated have a considerably higher frequency of type III than found in any of the other groups.

Among the 66 mongoloids no case of type III was observed, although the expectancy should be 16 or 17 cases. The absence of type III in the mongoloids may be a result of retardation of growth in the region of the head which normally brings forth this dominant character.

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TRANSPLACENTAL ISOIMMUNIZATION IN HORSES

THE publication of "Jaundiced-Foal-Producing Mares" by D. W. Bruner, F. E. Hull, P. R. Edwards, and E. R. Doll,[1] reveals the first, clear-cut example of isoimmunization across the placenta in a species other than man. The disease described in the foal resembles the hemolytic disease of erythroblastosis fetalis to a remarkable degree.

The cases reported by Bruner and associates comprise twelve mares which have produced one or more jaundiced foals. Each of these mares had produced three or more normal foals before giving birth to a jaundiced foal. The situation is different than that in humans in that the foals are born normal and become jaundiced from twelve to 96 hours after birth. The reasons for this difference are discussed below. Jaundice can be prevented by foster nursing the potentially jaundiced foal on a mare who has not borne a jaundiced colt. Jaundiced foals can be produced "artificially" by injecting a normal pregnant mare with the red blood cells of the stallion to which she has been bred.

In the report above cited the following criteria of transplacental isoimmunization are satisfied:

1. A presumably dominant hereditary property in the blood of the foal inherited from the stallion and absent in the mare.

2. The selective action of the antibody affecting the foal with blood properties

of the stallion and the absence of symptoms in the foal having the blood properties of the mare.

3. A preparatory phase of several pregnancies (at least three) with normal foals having the dominant property of the stallion which is required for immunization.

4. The influence of previous transfusions with blood of the stallion which induces isoimmunization far more rapidly.

5. The beneficial effects of transfusion to combat the anemia.

In only one important respect does the situation seem to be different from that in man. This refers to the absence of intra-uterine damage to the foal. In all instances, the foal seems to be normal at birth and develops jaundice within 12-96 hours after birth. From the standpoint of comparative anatomy, this is readily understandable because in ruminants and other species of animals characterized by certain types of placenta, the young acquire their natural immunity not by intra-uterine passage of maternal antibodies but rather by ingestion of colostrum and milk and absorption from the intestinal tract. In the light of this fact, the absence of intra-uterine damage lends still further proof to the striking analogy of jaundice in the foal and erythroblastosis fetalis in man.

From this it follows that all that is required for the specific therapy is (1) putting the foal on a nurse mare and (2) transfusion. In contrast to the condition in infants who should receive Rh negative blood, random horse blood may be used for the foal because maternal antibodies do not pass into the foal *in utero*. Once the foal has ingested large quantities of antibodies from its mare's milk, it would then seem advisable to select a blood of the same type as that of the mare.

Other Instances of Transplacental Isoimmunization

In a search of the literature made shortly after the description of the role of the Rh factor in erythroblastosis fetalis for similar instances in animals, two interesting papers (Robinson, Corner) were found.[2] "These investigators studied the causes of intra-uterine fetal deaths in pigs and ferrets, and infertility in horses. Robinson and Corner concluded that genetic and constitutional factors are probably responsible for a high incidence of fetal death. In the case of ferrets and pigs, these workers found normal fetuses alongside of dead macerated fetuses in a uterus which was free of detectable disease. Corner assumes the presence of genetic lethal factors analogous to those found in Drosophila and in the case of the yellow mouse studied by Cuenot, Castle and Little and Kirkham."[2] A more likely explanation, however, is isoimmunization and selective action on the particular fetus having the dominant property derived from the sire.

"There is no reason to assume that isoimmunization by the fetus should be limited to man. On the other hand, there is serologic evidence to indicate that isoimmunization by pregnancy should occur frequently in many animal species. It is a well-established fact, based experimentally upon the isoimmunization resulting from repeated transfusions, that the individuality of blood in many species is greater than among humans. This phenomenon seems to occur more readily in animals than in man. Furthermore, pregnancy offers certain conditions which are peculiarly favorable to isoimmunization, i.e., slow administration of the antigen over a long period."[2]

Different Placental Barriers

In a recent summary on the significance of the Rh factor, I have recently written as follows: "Undoubtedly, many examples will be found in veterinary medicine at least in those species which are characterized by a placenta which do not differ radically from that in man."[3] This brings up a very important question of the mechanism of transplacental isoimmunization in horses. In ruminants there are four layers of tissue cells which separate the fetal and maternal blood, in contrast to rodents, apes and man where

there is only a single layer of cells (in addition to the fetal endothelium) which forms the barrier. Accordingly, it becomes important to investigate more thoroughly the histology of the normal horse placenta in order to determine how the fetal red cell, which is a large formed element, finds its way into the maternal circulation in the absence of gross placental lesions.

It is very likely that, as in the case of the Rh factor, the particular horse blood factors involved are not present in a water-soluble form in body fluids such as saliva or gastric juice. As in the case of erythroblastosis, if the blood factors involved were present in water-soluble form, the maternal antibodies after absorption from the intestinal tract would then be neutralized by the excess of water-soluble antigenic material and no antibodies would be available to attack the red blood cells.[4]

It is hoped that Bruner and his coworkers will make careful hematological studies of the affected foal blood in order to establish the absolute barrier to the passage of maternal antibodies *in utero*. A higher than normal incidence of normoblasts found at birth, and even moderate degrees of anemia, may indicate the passage of at least smaller quantities of maternal antibodies.

I would like to question the remark of Bruner et al, "that abnormal placenta may contribute toward sensitizing some mares against certain types of red blood cells." So far as man is concerned, there is more and more evidence to indicate that in the course of every normal pregnancy, a few fetal red cells find their way into maternal circulation but in sufficient quantity to induce antibody formation in the mother. There is no need to assume the existence of gross lesions which would have to recur in each subsequent pregnancy.[5-9]

A very important factor in the mechanism of transplacental transfer of fetal blood in all species is the positive pressure supplied by the fetal heart and the very slow and sluggish circulation in the uterine sinuses and, as mentioned above, the minuteness of the dose of red cells required for antibody production. In all species there is a tremendous surface area of fetal tissue more or less exposed to maternal sinuses.[10, 11]

Genetic Factors Involved

It is hoped that Bruner and associates will soon publish their serologic findings in order to determine whether one or more than one factor is involved. It is remarkable that in man the Rh factor (D, in the Fischer-Race nomenclature) can explain more than 90% of all instances of isoimmunization, whether by transfusion or by transplacental transfer of fetal blood. Judging from our knowledge of the individuality of blood of horses and other species of animals as revealed by studies of isoimmunization, one may speculate that numerous factors will be involved. In any event, it now becomes important to identify the factors in terms of their specific antibodies, a supply of which should be kept on hand for the diagnosis of the condition in the foal and for selective breeding or artificial insemination in order to circumvent the neonatal morbidity attributable to transplacental isoimmunization. Accordingly, an important, powerful weapon is now available for the serologist who is in a position to cooperate in the selective breeding of horses.

One is now tempted to predict with a greater degree of certainty that selective fetal or neonatal morbidity will be found in all species of animals in which blood differences could be demonstrated with the aid of immune isoagglutinins or hemolysins resulting from cross-transfusions. The most surprising element in the contribution of Bruner et al is the fact that the four layers of tissue cells which separate fetal and maternal circulations constitute no absolute barrier for the fetal horse red cells.

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ADDENDUM: Dr. Mourant called my attention to identical findings in six cases studied by Coombs (*British Med. Jour. ii, 95, 1948*). In each case, evidence was presented that the foal's blood was coated with maternal antibodies. Similar findings were reported by Caroli and Bessis in the mule offspring of the horse-donkey cross (*Comp. rend. d.s. / Acad. d. Sciences 224,* 969, 1947).

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LOCATION OF THE PIROUETTE GENE IN *MUS MUSCULUS**

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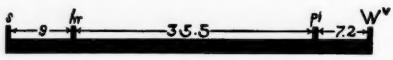
Pirouette,† a mutation causing choreic behavior and inability to respond to ordinary sound has been described.[2] Linkage studies revealed that the gene locus for this character was 7.2 units from the dominant spotting W^v locus on the third chromosome.[1] This chromosome was also known to carry hairless *hr* and recessive spotting *s* in the order *s, hr, W^v*. Backcross tests with hairless and pirouette show that the pirouette gene lies between hairless and dominant spotting. See Table I. Thus the four genes on the third chromosome are in the following order *s, hr, pi, W^v*. See Figure 6.

TABLE I.—Linkage data on hairless and pirouette genes

Cross	*HrPi hrpi* ♀ × *hrpi hrpi* ♂	
Non-crossover	No. Animals	
HrPi	20	
hrpi	18	38
Crossover		
hrPi	8	
Hrpi	13	21
Percentage Crossover		35.5



GENES ON THE THIRD CHROMOSOME OF THE MOUSE

Figure 6

The genes now located on this chromosome comprise the following: Recessive spotting (*s*), *hairless* (*hr*), pirouette (*pi*), and viable dominant spotting (W^v).

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*This work has been aided by grants to the Roscoe B. Jackson Memorial Laboratory by the Commonwealth Fund, the Anna Fuller Fund, Jane Coffin Childs Memorial Fund for Medical Research, and the National Advisory Cancer Council.

†This linkage study was terminated by the fire which destroyed the Jackson Laboratory October 23, 1947. The gene, however, has been recovered and is now available for further studies.

HEREDITARY MICROPHTHALMIA AND OTHER EYE DEFECTS IN PIGEONS

W. F. HOLLANDER*

AMONG several pairs of his "Giant Homer" pigeons, Mr. Carl F. Graefe of Cuyahoga Falls, Ohio, noted that one repeatedly produced small-eyed blind offspring. These were fed by the parents usually beyond weaning age, but had to be killed and were preserved in formalin for study. These specimens together with the living parents and several related individuals were sent to me in 1944.

Since then I have progeny-tested these birds and their descendants rather systematically. The microphthalmic condition has recurred frequently and is readily recognized at or before hatching. The affected young are totally blind, with typical nystagmus. I have not attempted to teach them to feed themselves at weaning age; no information on their possible fertility is available therefore. However, anatomically they are perfectly normal males and females.

An analysis of the pedigrees and breeding data clearly indicate that the trait is a simple Mendelian recessive, and not sex-linked. It is impossible to recognize any abnormality in the eyes of the parents. The symbol *mi* is proposed for the inherited factor.

The microphthalmia has segregated at random with two other autosomal factors—recessive red plumage color and grizzle plumage color. At present there is no suggestion of linkage.

A few of the microphthalmic squabs have grown vigorously, but more often there has been retarded growth. Also it will be noted that the proportion of microphthalmics among the advanced embryos which died is high (Table 1). Possibly the moderate inbreeding of the stock is responsible for part of the inferiority, but this gene may itself well reduce vigor.

Dissection of the eyes of the blind squabs shows that though the eyeball is only about half normal size it is constructed very much like the normal. The most obvious differences are the greatly thickened choroid layer, and the relatively small chamber of the vitreous humor. Apparently the retina is defective also, but it is present. Further study of the histology and embryonic development of such eyes will be reported elsewhere. Whether the blindness is entirely a matter of abnormality within the eyeball is not yet known; the eyelid opening is small and the eyelids are not functional, and would obstruct vision.

In addition to the above type of microphthalmia there have been several other cases among pigeons. Levi[6] reported on a female white Carneau apparently lacking eyes. This specimen was able to reproduce but the condition appeared to be non-genetic. The bird was killed in 1944. Rudiments of eyes about 2 mm. thick were found.

A microphthalmic Racing Homer squab (small eyes) was bred by Mr. Earl A. Klotz (now of Cleveland, Ohio), but the inheritance was not investigated. Another and very remarkable example in the Racing Homer breed was bred in the Spring of 1945 by Mr. Wiley Sholar in North Carolina, and transmitted to me by Mr. W. M. Levi. This specimen (AU45L 7912) has eyes about half normal size, but there is some vision. The eyelids are small but functional. Also, the plumage is defective, being short and poorly webbed. The bird cannot fly on this account. And finally there is a fairly well developed web of skin between the center and outer toes of the left foot. Although aggressive and sexually active as a male, the bird has entirely failed to fertilize eggs. Possibly inability to copulate normally

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is responsible, and artificial insemination may be successful.

It seems worthwhile at this point briefly to review the studies of pigeons' eyes. Gundlach and associates[1] have investigated the structure of the eye of the normal Homer pigeon, especially with reference to accommodation. Iris pig-

SMALL-EYE ADULT

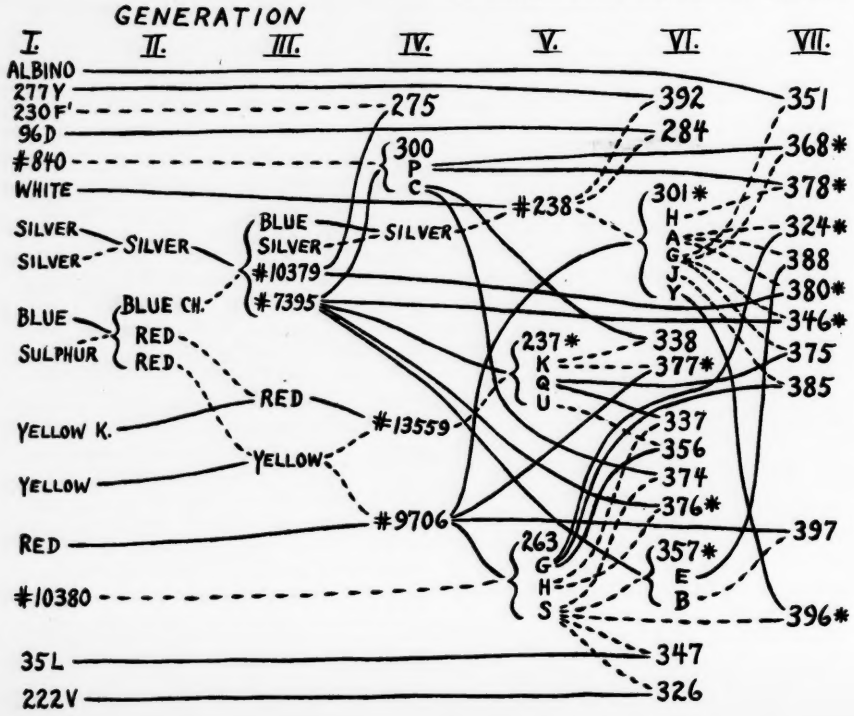
Figure 7

Microphthalmic male AU 45 L 7912 of the Racing Homer breed. Note small eyes, defective plumage, and web between center and outer toes of left foot.

PEDIGREE NETWORK OF MICROPHTHALMIC STRAIN

Figure 8

Read from left to right, from oldest to youngest. Solid lines are from sires, dash lines from dams. The sign # before a number indicates an original bird given to the author. Other numbers are mating numbers (= sibships); letters represent individuals. An asterisk following a mating number signifies that microphthalmic offspring were produced.



MICROPHTHALMIC AND NORMAL SQUABS

Figure 9

A—Heads of two squabs just hatched to show the difference of appearance of normal (left) and microphthalmic eyes. *B*—Heads of squabs practically full sized (about a month old). The eyelids and skin around the eyes have been removed to show size differences of the eyeball. Their differences in shape of head are incidental breed differences. (About natural size.)

TABLE I. A tabulation of the results of matings which produced microphthalmic squabs.

MATING #	GENERATION	TOTAL EGGS	INFERTILE	NO RECORD – LOST, BROKEN	DEAD EMBRYO EARLY	NOT HATCHED EMBRYO 8+ DAYS		HATCHED	
						mi.	NOR.	mi.	NOR.
237	V	27	6	2	3	1	5	5	5
301	VI	24	1	1	1		2	2	17
324	VII	8		2		1	1	1	3
346	VII	2				1	1		
357	VI	8	2				1	1	4
368	VII	2				1			1
376	VI	10	4	2		1	1		2
377	VI	6				2	2		2
378	VII	4	1			1			2
380	VII	6	2			1	2	1	
396	VII	8	1			1	2	2	2
TOTAL		105	17	7	4	10	17	12	38
						22 mi. : 55 NOR.			
EXPECTATION						19 : 58			

TABLE II. A tabulation of the results of outcrosses and other matings involving pigeons which produced microphthalmic offspring in matings given in Table I.

MATING #	GENERATION	TOTAL EGGS	INFERTILE	NO RECORD – LOST, BROKEN	DEAD EMBRYO EARLY	NOT HATCHED EMBRYO 8+ DAYS		HATCHED	
						mi.	NOR.	mi.	NOR.
263	V	18		6					12
275	IV	24	15				2		7
284	VI	2							2
300	IV	16	2	4			1		9
326	VI	4							4
337	VI	10		3			1		6
338	VI	12		2	1		1		8
347	VI	2							2
351	VII	4							4
356	VI	10		2			2		6
374	VI	6		3			1		2
375	VII	14		2			3		9
385	VII	12		2			1		9
388	VII	14	1	6	1		5		1
392	VI	8							8
397	VII	12							12
TOTAL		168	18	30	2	0	17	0	101

mentation and its variations and their inheritance have been studied by several persons, and the subject has already been reviewed by Hollander and Owen.[4] In addition albino pigeons have been studied by Lienhart[7] and Hollander (unpublished). These pigeons are visually handicapped like albinos in other species, and show marked nystagmus.

Another hereditary factor affecting plumage, skin, and eyes, the dominant sex-linked "almond" color, has been known for a long time to be associated with a sporadic defect known as "bladder eye." Wriedt and Christie[8] also found coloboma of the iris in connection with this color factor, but the "bladder-eye" condition seemed to them usually to be the result of combination of almond with another factor, "dilution." The inheritance of these sex-linked factors has been reviewed by Hollander.[3] Further study indicates that homozygous almond is almost always associated with defective (irregular) iris structure, and that "bladder-eye" at least sometimes involves total absence of the lens.

Lethal polydactyly, reported by Hollander and Levi,[5] was noted to involve also poor vision in the longest-lived specimen. Subsequently the vision of additional specimens has been tested and found defective in all cases, almost amounting to blindness. Dissection of the eyes of such polydactyls has revealed no obvious abnormality.

A recessive hereditary eye defect, which has been referred to by its symptoms, "clumsy," was reported by Hollander.[2] This appears to be a rather minor defect of the retina, but the birds are seriously handicapped.

From this survey one can see that the pigeon is becoming a rather promising laboratory medium for the study of eye morphogenesis.

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Death Takes A Selective Holiday

The shift in distribution of our population from youth to middle and old age, largely a result of success in learning how to combat infections, has brought medical research to a new frontier of intensive exploration. The degenerative diseases are dominant in the foreground, and it is already evident that malnutrition is a major factor in their origin and course. The urgency of research in this area scarcely needs emphasis. For example, present data indicate that:

(1) One-half of the babies born in 1940 probably will die of degenerative disease of the circulatory system and kidneys.

(2) Chronic heart disease, cancer, hardening of the arteries, cerebral hemorrhage, chronic nephritis and diabetes rank among the first ten causes of deaths in the United States and account for 60 per cent of the total.

(3) Cerebral hardening of the arteries accounts for a large fraction of the patients admitted to mental hospitals, of whom 30 per cent are sixty years or more of age.

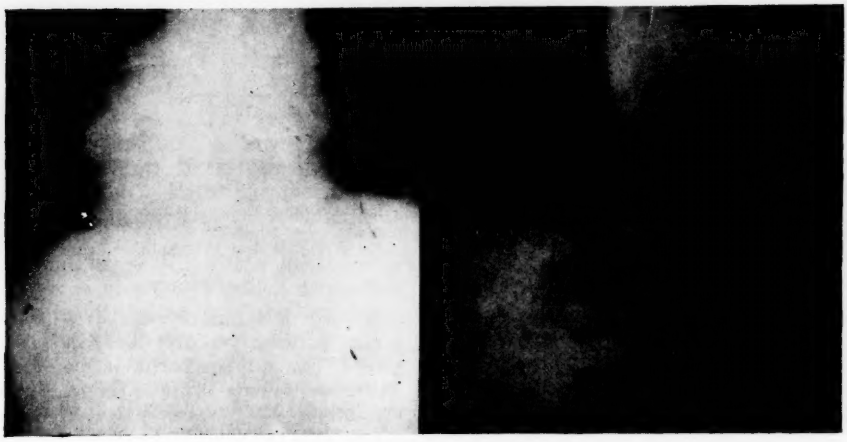
(4) More than a fourth of the 6.8 million people with rheumatism have the degenerative form of arthritis.

(5) The average life expectancy is now 65.1 years for white males and 70.3 years for white females. For the non-white population, the corresponding figures are 57.5 and 61.0 years, respectively. In other words, children born today have a life expectancy of about eighteen years beyond the age that could be claimed for their grandparents. Good nutrition can make a contribution to those added eighteen years being years of vigorous living, not just survival.

Charles Glen King,
Director of the Nutrition Foundation.

CONCORDANT SITUS INVERSUS IN DIZYGOTIC TWINS

JOHAN TORGERSEN
Anatomical Institute, University of Oslo, Norway



X-RAY OF CHEST OF TWINS
Figure 11

A shows chest of twin J with heart to the right, liver to the left. *B* shows the chest of twin M with heart to the left and stomach to the right. Possibly other idiosyncracies shared by the twins are associated with the inversion.

IN the twin brothers J. and M. I found complete situs inversus in J., situs inversus of the abdominal organs in M.

As seen in the photograph (Figure 12) they look rather different.

The mother was 36 at the birth of the twins. The father is fair with a short, broad face like M.; the mother dark with a long face like J.

There are many twins in the family of the father, probably all dizygotic. (See Figure 13). Twins have not occurred in the family of the mother. Symptoms of hay fever did not occur among the siblings.

It is highly improbable that the coincidence of situs inversus in these twins depends on chance or that the many twins in the father's family are coincidental.

The concordance of the twins with respect to the extreme variations of the spine and the similar allergy suggests the possibility that there may be connection with the processes causing the inversion.

Concordant as well as discordant situs inversus in monozygotic twins has been

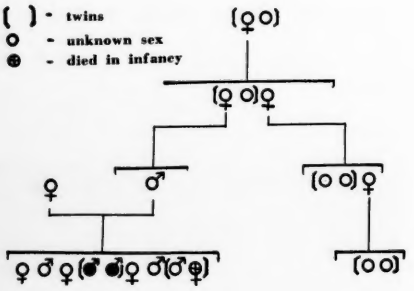
TABLE I.—Comparison of twins M. and J.

M.	J.
Farmer.	Farmer.
A paresis of the right leg after poliomyelitis.	
Height: 171 cm.	180 cm.
Length of the skull: 19.3 "	19.5 "
Breadth " " " : 15.3 "	15.0 "
Min. front. breadth: 10.6 "	10.1 "
Length of the nose: 5.4 "	5.8 "
Breadth " " " : 3.6 "	3.1 "
Bigonial width: 10.6 "	10.3 "
Eye colour (Martin): 3	5
Hair " " : U.	P.
The texture of hair and skin is different.	
Stomach on the right.	Complete situs inversus.
Liver and duodenum on the left.	
5 lumbar vertebrae plus a lumbosacral vertebra.	5 lumbar vertebrae plus a thoracolumbar vertebra.
Frontal sinus average size.	Frontal sinus average size.
Marked symptoms of hay-fever.	Very slight symptoms of hayfever.
Righthanded.	Righthanded.
Extrovert and easygoing.	Introvert and sensitive.

TWINS J AND M
Figure 12

The twins do not look particularly alike, and there are other evidences that they are dizygotic. Hence the inversion of their internal organs cannot be attributed to mirror imaging.

observed several times. This observation of situs inversus, and particularly of the discordant manifestation, in twins which most probably are dizygotic, may be of interest to theorists on twinning and asymmetry. This is also the case with the probably hereditary disposition to twin birth in the family of the father.



HIGH TWINNING ANCESTRY
Figure 13

Twins J and M are identified by the black symbols. In addition to twin siblings, the father's mother and grandmother were twins.

Summary

Record of a unique case of situs inversus in twins, probably dizygotic complete in one of them, partial in the other. There were many twins in the family of the father.

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TYPE-KNAPPING AND THE OHIO OCTUPLETS

TO THE EDITOR:

Dr. Newman's query in your July issue about the verification of extravagant multiple births in this country interested me. It is very easy to refute the Bradlee case because this was most expertly done by Dr. G. H. Parker in *Science* (the new series) Volume 41, No. 1061, April 30, 1915. Dr. Parker calls his article "A Spurious Case of Multiple Human Births." It appears that Dr. Parker was justly skeptical of the survival of any of the supposed brood. In turn he wrote a letter to the clerk of the Trumble County Court in Warren, Ohio, where the event was supposed to have occurred, and the clerk in turn did some fancy sleuthing.

It appears that the whole event was a complete figment of the imagination. A rebuffed suitor of a Eunice Mowery who is stated to have littered the brood, tried to get even with the said lady by spreading the calumny. By chance he was a printer and stole into the premises of the local paper, removed a block of type from the next morning's issue and reinserted in its place the squib reported in Dr. Newman's article. He ran off some twenty or thirty copies and sent them to the more important Eastern dailies. He then replaced the original block of type so that the local paper carried no report of it. Apparently the scheme worked because the exciting news was printed in many newspapers and even found itself on the pages of the staid Boston Medical and Surgical Journal of that year. Thus, thanks to Dr. Parker we can dispose of these human octuplets.

I investigated quite carefully other amazing instances of large human litters and haven't found a case of even so much as a single quintuplet surviving, until the marvelous Dionnes came along. There have been several authentic cases of sextuplet births but in no instance has there been proved temporary survival of even one infant. The only case of septuplets which has any semblance of truth is that of the famous Hameln septuplets, who may or may not have been born in 1600.

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THE INCIDENCE AND PROBABLE INHERITANCE OF "SMELL BLINDNESS"

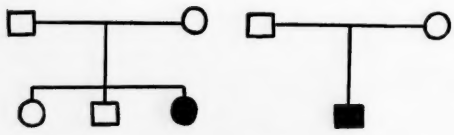
To Normal Butyl Mercaptan

PAUL M. PATTERSON AND BARBARA A. LAUDER
Hollins College, Virginia

THE purpose in this study has been to follow up a statement made by Gates[4] (p. 1066) that some people are unable to detect the odor of normal butyl mercaptan, which closely resembles the odor produced by the skunk. Our aim has been to determine the proportion of people who cannot detect this odor and to show how this character might be inherited.

Previous work on smell "blindness" has not been limited to normal butyl mercaptan. Blakeslee[1] found that there are differences among people in the ability to smell certain *Verbena* flowers. A third of the people he tested could smell only pink verbenas while the rest could smell only the red. Glaser[5] studied a single family in Russia where the mother and grandfather were completely anosmic; two sons were unable to smell any solutions containing high percentages of alcohol and did not have normal reactions to ether, chloroform, or pepper; one son had only slight difficulty in detecting odors; two daughters were normal. He claimed that it was caused by a sex-linked factor. Stockard[8] doubted the accuracy of Glaser's report; he pointed out that the occurrence of rhinoscleroma in certain parts of Russia may cause anosmia. According to his report, there are many persons with otherwise normal ability to detect odors who cannot smell some of the more pungent substances. He claimed that anosmia was not sufficiently frequent in men to be considered sex-linked. Blakeslee and Salmon[3] made further tests with pink and red *Verbena* and, in addition to the differences earlier noted, indicated that there was considerable variation in the description people gave to the odors detected. The authors also indicated that many individuals are unable to smell certain unspecified types of odors. Nyssen, Helsmoortel and Thienpont[7] summarized various opinions about the inheritance of anosmia. They cited Collet who described a case of a mother and son, both anosmic, where sex-linked inheritance was inferred. Davenport had claimed that anosmia was transmitted recessively and was sex-linked. Alikhan was also reported to have shown that the transmission was sex-linked in that the female transmitted the character to the male and female children equally. Nyssen et al. tested three members of a family of twenty-eight, all of low intelligence, five of whom were reported anosmic from birth. The three tested could not smell thymol, heliotrope, methyl salicylate, iodoform, caproic acid, or skatole. The authors claimed that this anosmia was transmitted by males and females for several generations. Blakeslee[2] tested smell reactions to two types of *Freesia*, one with a strong odor (for most people) and one with a weak odor. He asked people to determine whether the odor of each type was weak, medium, or strong, pleasant or unpleasant, and found little consistency among them in their answers. He further suggested that complete anosmia was characterized by its appearance after twenty-five years of age, but pointed out that cases of anosmia from birth have been reported. Mainland[6] reported that anosmia in a family she investigated behaved as if caused by a single dominant autosomal gene.

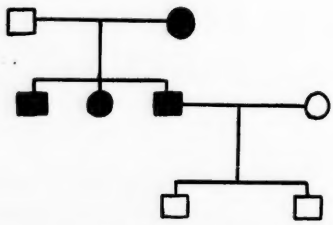
In this investigation, a concentration of 0.0075% normal butyl mercaptan dissolved in 90% methyl alcohol was used.



NORMAL PARENTS, AFFECTED CHILDREN

Figure 13

If this type of smell blindness is hereditary, it would appear from this very meager information to be inherited as a recessive characteristic.



INHERITANCE OF DELAYED ANOSMIA

Figure 14

In this family, loss of ability to smell has come on in middle age. Dominant inheritance is suggested. It is found in this survey that smell blindness is extremely rare in the sample of population studied.

Methyl alcohol was selected as a solvent since it is relatively odorless. Even at this dilution, the mercaptan was decidedly pungent or obnoxious to the average person. It was distributed in plastic screw-cap, one-ounce bottles.

Four thousand thirty persons have been tested through the cooperation of teachers and students in high schools in Roanoke and Botetourt Counties and at Roanoke and Hollins Colleges.* Volunteer students were given bottles of the mercaptan solution and were asked to test their families and to record their reactions on provided forms. Nearly all those who were reported negative to the first test have been visited and tested with solutions of normal butyl mercaptan five and ten times as strong as the original testing solution. At the same time they were further tested with one characteristic odor at or near each of the six corners of the smell prism as arranged by Henning (Woodworth,[9] p. 486-487). The types of odors and the examples used in this follow-up test were: fragrant—commercial vanilla extract, spicy—oil of cloves, resinous—oil of eucalyptus, etherial — oil of orange, burned—tar, and putrid—hydrogen sulfide where aqueous solutions were prepared by the hydrolysis of sodium sulfide. Persons were carefully questioned with respect to diseases or accidents which might affect the olfactory sense.

Four types of deficiency in olfactory perception have been found (see Table I). One type is characterized by inability to smell normal butyl mercaptan with no difficulty in detecting the majority of the other odors used for the follow-up test. There were four such individuals found. Retests in this group revealed several irregularities in olfactory perception. Two individuals were unable to

TABLE I. Smell Blindness Types

	Typical smell "blindness" to n-butyl mercaptan	Partial smell "blindness" to n-butyl mercaptan	Delayed anosmia	Smell "blindness" due to accident or disease
Percent of total	0.099%	0.050%	0.099%	0.171%
Number of persons	4	2	4	7

TABLE II. Results of Follow-up Tests of the First Two Groups

Normal Butyl Mercaptan								
0.0075%	0.0375%	0.075%	Oil of Eucalyptus	H_2S	Tar	Oil of Orange	Oil of Cloves	Vanilla Extract
—	—	—	—	+	+	+	+	+
—	—	—	—	+	+	+	+	+
—	—	—	+	+	+	±	+	+
—	—	—	+	+	+	+	+	+
—	±	±	+	+	+	+	+	+
—	±	±	+	±	±	+	+	+

*We acknowledge our indebtedness to the following for their cooperation in distributing bottles of normal butyl mercaptan to students: the principals and science teachers of: Andrew Lewis High School, Salem, Va.; Jefferson High School, Roanoke, Va.; Lee Junior High School, Roanoke, Va.; Troutville High School, Troutville, Va.; William Byrd High School, Vinton, Va.; William Fleming High School, Roanoke, Va.; and the Biology Department of Roanoke College, Salem, Va.

detect the odor of pure oil of eucalyptus which seemed very strong to the others, and another found that pure oil of orange had only a faint odor. Aside from this, all four had normal sense of smell for the materials tested except normal butyl mercaptan. If it may be assumed that this type of smell "blindness" is inherited, it would appear to be a recessive character because children whose parents are normal have been affected (see Figure 13). The data are insufficient to indicate whether this is a single mendelian recessive or whether the inheritance pattern is more complex.

A second group consists of two individuals who were unable to smell the 0.0075% solution of normal butyl mercaptan but who could detect a slight odor from the stronger solutions of the mercaptan. One of these persons had no difficulty in smelling the other test solutions; the other, who suffers from chronic sinusitis, could detect only mild odors from hydrogen sulfide and tar, but seemed to smell the others normally (see Table II).

A third type of smell deficiency was found in one family. While only one member of this family, a middle-aged anosmic man, was interviewed, there was every indication that his account of his mother, sister, and brother was substantially accurate. Anosmia appeared in the mother and the three children of this family at middle age. There had been no recallable incidence of disease or accident which might affect the sense of smell. The man interviewed has two sons of school age who are normal for olfactory sense. It would appear that this type of anosmia is inherited as a dominant character with delayed expression (see Figure 14).

The fourth group was characterized by reduced sensitivity or complete anosmia as a result of accidents, diseases such as chronic sinusitis, or nasal operations. The seven persons in this category are not described since their anosmia is not considered to have a genetical basis.

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FROM THE CONGRESS ABSTRACTS

Mustard Gas Mutations

THE starting point for the genetical work with mustard gas was the similarity between mustard gas burns and X-ray burns; this suggested the possibility of a nuclear action of mustard gas. Experiments on Drosophila have fully borne out this assumption. Treatment with suitable doses of mustard gas sterilizes males and females through interference with gametogenesis and through dominant lethality of the zygotes. The treatment induces sex-linked lethals, minute deficiencies and visible mutations in frequencies which compare well with those produced by high-energy radiation. No mutations are produced in untreated sperm which through fertilization has been introduced into treated ova. When treated males are mated to a succession of virgin females, mutation rate usually rises in successive broods up to a peak at about 6 days after treatment. * * *

Large re-arrangements are also produced in appreciable numbers, but their frequency in the progeny of treated males falls markedly short of what would be expected from an X-ray dose producing the same percentage of sex-linked lethals.

The most outstanding peculiarity of chemical mutagenic action appears to be the induction of localized instabilities which subsequently give rise to a mutation or chromosome break. * * * Allyl isothiocyanate occurs naturally in Brassica plants. The possibility has to be considered that natural chemical mutagens have played a role in adapting mutability to the requirements of evolution.

A number of continental and American

workers have reported mutagenic activity of substances not belonging to the mustard group. These results will be briefly discussed.

— CHARLOTTE AUERBACH

Evaluating Heredity-Environment

TOGETHER with a number of coworkers the interaction between environment and heredity in the determination of production level is studied through the use of identical cattle twins. These studies include rate of growth of body weight and body measurements, milk yield and milk calories. Concurrently a close study is made of the constituents of the milk. As a result of these and related studies it is found that heredity plays a very important role, usually by far outweighing the effect of environmental influences.

— G. BONNIER

Industrial Melanism

THE spread of dark forms of the Lepidoptera in industrial areas during the last hundred years constituted the greatest evolutionary change ever witnessed. Many species, belonging to widely different groups, are affected. They have in common a cryptic pattern and the habit of resting in exposed positions. Dark varieties have been observed in many species which have never become industrial melanics. In no known instance have they become common if inherited as simple recessives. The great majority of industrial melanics are autosomal dominants; a few are heterozygous forms or are multifactorial. Selection favouring blacking specimens in areas where the vegetation is darkened by soot is not a sufficient explanation of industrial melanism, for it does not cover the facts. It is now known that some, and inferred that all, industrial melanics differ physiologically as well as in colour from the normal form, being hardier. Also they have within recent times spread in a few areas which are not darkened by soot. It is suggested that species have used those genes which have conferred the greatest physiological advantage unless this be outweighed by other and disadvantageous effects, such as the obliteration of cryptic colour-pattern by excess melanin formation. * * *

E. B. FORD

Mutation in Man

HUMAN mutation rates were first estimated by Penrose and Haldane in 1935. Since then they have been estimated, often with greater accuracy, by workers in Kemp's Institute in Copenhagen, by Nachtsheim and Pätau, and by Neel. The mutation frequency of dominant genes can be estimated directly, by finding the number of mutants born of normal parents, and indirectly by finding the frequency of the condition and the lowering of fitness which it causes, and calculating the mutation rate in equilibrium with the adverse selection. Mörch showed that these two methods agree in the case of chondrodystrophy. The frequency of sex-linked recessives can only be estimated from the equilibrium, though some pedigrees show clearly that mutation occurs. No estimates can be made in the case of autosomal recessives, as we do not know the fitness of the heterozygotes, and, owing to the recent relaxation of inbreeding in civilized countries, populations are not in equilibrium. * * *

The most mutable human genes are somewhat less stable per generation than those of Drosophila, but far stabler per day. Most human mutations may well be due to particles of high energy. The mutagenic effect of radiation from atomic fission can be overestimated, since the lethal dose for man is much less than that for Drosophila. Thus an atomic bomb explosion is likely to cause many more deaths immediately than from lethal genes in later generations.

A plea is made for further research on this topic, and methods of research are suggested.

— J. B. S. HALDANE.

Heritability of Quantitative Characters

METHODS of estimating heritability are all based on measuring the degree of likeness between individuals which are related in various ways. Resemblance may be measured either by comparing the variances between individuals related in various ways or as a correlation. That part of the resemblance caused by environmental correlations needs to be discounted before interpreting resemblance as heritability. Also allowance is to be made for non-randomness in the mating system. * * *

Data for a wide variety of characters of considerable economic importance show heritabilities of intra-herd individual phenotypic differences generally of the order of .10 to .30 or more. This includes only the genic variance and traces of the epistatic variance within herds. Heritabilities this high seem a bit inconsistent with breed improvement being slow while intense selection is practiced. Plausible or at least possible explanations include the following: (1) Breeders may not actually practice as intense selection as we think, (2) the breed means may actually be changing more rapidly than we think, (3) the epistatic variance may be a large fraction of the heritability measured, and (4) genetic correlations between the various desirable characters may be more strongly negative than we generally suppose. If the latter is so, the progress made by selecting directly for a character at one stage may be largely cancelled by the indirect selection against it when selecting for some other character at another stage. Statistically and practically this leads to much the same results as overdominance.

— J. L. LUSH

Allopolyploids in Evolution

THE family Gramineae contains an array of polyploid types showing all intergraduations between strict autopolyploidy and complete allopolyploidy. The former is represented by several artificial autopolyploids, including the one produced artificially from *Ehrharta erecta,* which has been established in nature for five years. Intervarietal autopolyploids are exemplified by *Dactylis glomerata,* and by one involving two races of *Bromus stamineus,* which has shown exceptional vigor. Segmental or incomplete allopolyploids, derived from hybridization between different ecospecies belonging to the same cenospecies, have been produced in the genera *Bromus* and *Melica,* and in the tribe Hordeae. Typical or complete allopolyploids, derived from hybridization between different cenospecies, are well known in the family both as natural species and artificial productions. * * *

The eventual success of the new allopolyploids depends largely on maintenance of the original fertility, or increase of fertility in progeny of partially sterile "raw" allopolyploids. This is related partly to the pairing behavior of the chromosomes, and partly to other factors affecting regularity of meiosis, as well as to sterility factors of an unknown nature. Artificial allopolyploids produced by treatment with colchicine are usually rather fertile in the first generation, but in many of them this fertility later declines for two or three generations, after which selection for increased fertility is possible in some lines. The cytological basis of these changes in fertility, and their significance in relation to concepts of auto- and alloploidy, are discussed.

— G. L. STEBBINS

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Hemophilia Is Democratic, Too!

TO THE EDITOR:

If the oft-told tale of hemophilia in descendants of Queen Victoria is to be re-hashed again in the near future, it is to be hoped that the rehashers will refrain (1) from designating it as "the royal disease," (2) from dragging into their discussion living persons who have no chance whatever of carrying the gene (unless by new mutation), and (3) from public speculation, in the worst Winchellian manner, about coming events that may have cast their shadows before (*J. Hered. April,* 1948, pp. 113-116).

Hemophilia might more appropriately be labelled as Philadelphia disease, because it was there first described as a specific clinical entity, or perhaps because of the Quaker lady who bequeathed it to the seven generations of her descendants in the pedigree compiled by Davidson and McQuarrie. It might be called the American disease, because the first extensive pedigree of it was reported in this country. It might even be designated as Illinois disease because in nine years no fewer than 98 hemophilic patients were encountered at a single hospital there by Dr. Caroll Birch.

Taking a more international view, hemophilia might be called Black Forest disease because there are several pedigrees of it from that area, or Danish disease because of the 205 cases in 63 families uncovered by Andreassen's valuable study of it in Denmark.

With a little more publicity on the universal distribution of hemophilia in the human race we might eventually dispel the prevalent misconception illustrated by the student—obviously no believer in monarchical government—who informed the writer in her examination paper that "Hemophilia is a dangerous sex-linked characteristic, even though limited to a great extent to the royal families of Europe."

The occurrence of hemophilia in some descendants of Queen Victoria shows that mutations are no respecters of persons. Must those who write about them follow suit? At any rate, if anyone wishes to designate poliomyelitis as "presidential disease" and to write about the possibility of its appearance in children yet unborn, it is to be hoped that such writings will not find a place in the JOURNAL OF HEREDITY. Even if it did have yellow covers for twenty-five years, it now has white ones.

In the interests of scientific accuracy, it is perhaps justifiable to point out that when any daughter of a non-hemophilic father is married to a non-hemophilic husband, the only possible source (barring new mutation) of a gene for hemophilia that might be transmitted to their offspring is to be sought in the lady's maternal ancestors. These were about the only relatives not mentioned in the discussion of such a case by Iltis cited above.

F. B. HUTT

Cornell University
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MOUSE GENETICS NEWS

Number 2

L. W. Law*

MOUSE Genetic News, Number 1, was compiled, mimeographed and distributed by Dr. G. D. Snell of the Roscoe B. Jackson Memorial Laboratory. This first number was distributed in November 1941. The demand for copies by investigators working with inbred strains and mutant stocks of *Mus musculus* was so great that the initial number was remimeographed in November 1945. Since that time new mutations and new linkages have been discovered, some important inbred strains of mice have become lost, other important inbred strains and sub-lines of existing strains have been developed. It seemed imperative therefore to compile a list of all known mutants with known linkage groups and a list of all known inbred strains of mice. It is hoped that problems concerning nomenclature, genetic symbols, etc., will in this manner be raised and that the listing of inbred strains of mice maintained in various laboratories will make them more readily available for further research in the fields of cancer, experimental medicine, psychology, genetics and allied fields. The ever-increasing number of scientists whose work has a direct or indirect bearing on the genetic basis of differences between mice and the ever-increasing cognizance that genetically homogeneous strains of mice are a necessity in their work have led to numerous inquiries and a tremendous demand for these experimental animals.

Many new mutant genes have been added to the list published in 1941 and adopted by the Committee on Mouse Genetics Nomenclature (Dunn, Grüneberg and Snell, *J. Hered.* 31:505-506). Minor changes have in some cases been made in the symbols or descriptive terminology of mutants to conform with the rules adopted by the Committee on Mouse Genetics Nomenclature. It is hoped that this Committee will be reconstructed so that such matters as nomenclature, assigning symbols to mutants, listing of specific mutants, etc., can be properly coordinated.

In the lists of mutations are given: 1) the name of the mutation, 2) the symbol of the mutation which for most of the mutants listed has been adopted by the Committee on Mouse Genetics Nomenclature and for more recent mutants the recommended symbol is given, 3) the linkage group, where known and 4) at least one reference. The reference may not be in each case the original reference. An attempt has been made to give articles best describing the mutation, a reference to linkage and where possible a recent reference.

For some of the mutants listed, complete data indicating a separate mutation from phenotypically similar mutations are not available, and in some cases more data are necessary to prove simple segregation. Numerous characters, structural and physiological in nature are known to be inherited, e.g. pig-tail, posterior duplication, headdot, ectromelia, etc. At present, however, they cannot be classed as simple recessives or dominants.

The list of inbred strains given herein is by no means complete and it is suggested that any omissions be called to the attention of the Editor. It is to be remembered that the characteristics listed for the most widely developed and used strains, e.g. *dba*, C_3H, *A*, etc., are usually those listed from a single laboratory and may not fit the description of sublines developed in other laboratories.

Dr. G. D. Snell and Dr. W. E. Hes-

*National Cancer Institute, National Institute of Health, Bethesda, Maryland.

ton have carefully reviewed the material and offered constructive advice for its presentation. The form of the presentation is essentially that used in the first issue and credit for the groundwork in making such a survey is due Dr. G .D. Snell of the Roscoe B. Jackson Memorial Laboratory.

Rules for Assigning Symbols to Mutations

The following rules for assigning symbols to mutations were adopted by the Committee on Mouse Genetics Nomenclature (Dunn, Grüneberg and Snell, *J. Hered.* 31:505-506).

1. *The initial letter* of the mutant symbol shall be the same as the initial letter of the mutant gene, e.g., *d* for dilution.

2. *Additional letters* shall be added to the initial letter if necessary to distinguish it from symbols already in use. These shall be, preferably, those immediately following the initial letter, or suggestive letters, especially consonants, from the rest of the name, e.g., *dw* for dwarf, *ac* for absence of corpus callosum.

3. *Recessive mutations* shall be indicated by the use of a small initial letter for the symbol of the mutant gene, the type allele being distinguished by a capital letter, e.g., *a* for non-agouti, *A* for agouti.

4. *Dominant mutations* shall be indicated by the use of a capital initial letter for the symbol of the mutant gene, the type allele being distinguished by a small initial letter, e.g., *Re* for rex, *re* for the type allele of rex.

5. *The wild type* may also be represented by a + rather than by a letter when this is more convenient, or by a small letter with a+ superscript, e.g., + or t^+ for the type allele of *T*, + or a^+ for the type allele of *a*.

6. *Multiple alleles* (except lethals) shall be indicated by the use of superscripts (always small letters, never capitals) added to the symbol of the original mutant type, e.g., c^{ch} for chinchilla. It is suggested that the letter selected be the initial letter of the name of the mutation, e.g., A^y for the yellow allele of agouti. The initial letter of the name of the discoverer may also be used. Lethal alleles in a multiple series may be indicated by the use of superscript numerals, e.g., t^0, t^1, t^2 and t^3 for the lethal alleles of brachyury.

7. *Mimics*, i.e., mutants of similar phenotype but different location, shall be indicated either by entirely different names and symbols (e.g., *ln* for leaden and *d* for dilution) or by the same name and symbol with the addition of distinguishing numbers (e.g., *wa-1* for waved-1 and *wa-2* for waved-2). The latter procedure is not recommended.

8. *In published articles* in American journals in which symbols are used, the symbols should be set in italics.

Recognized and Named Mutations

Mutation*	Recommended Symbol	Linkage Group	References
agouti series (agouti, lethal yellow, light-bellied agouti, black-and-tan, non agouti)	$A^y A^w + a^t a$	V	17, 26, 66
absence of corpus callosum	*ac*		55, 58
anemia	*an*	VIII	49
brown	*b*	VIII	66
belted	*bt*	VI	74
albino series (color, intense chinchilla, chinchilla, extreme dilution, albinism)	$+c^l c^{ch} c^e c$	I	21, 36a, 36b, 85
caracul	*Ca*	VI	5, 13
congenital hydrocephalus	*ch*		46
dilution	*d*	II	3
dwarf	*dw*		79
eyeless-1	*ey-1*		8, 9
eyeless-2	*ey-2*		8, 9
flexed tail	*f*		45, 52, 53
fidget	*fi*		46
fused	*Fu*	IX	34, 76
fuzzy	*fz*		89
grey-lethal	*gl*		42
histocompatibility-2	*H-2*	IX	41
hairless series (type, hairless, rhino)	$+hr\ hr^{rh}$	III	14, 51, 80
hypotrichosis juvenilis	*hj*		63
hydrocephalus-1	*hy-1*	V	11
hydrocephalus-2	*hy-2*		92
hydrocephalus-3	*hy-3*		46
jittery†	*j*	X	20, 82
jerker	*je*	XII	38, 44
kinky tail	*Ki*	IX	6, 34
kreisler	*kr*	V	49
leaden	*ln*		73
luxate	*lx*		75
misty	*m*	VIII	86, 87
microphthalmia	*mi*		49
myelencephalic blebs	*my*		67
naked	*N*	VI	13, 18, 61
open eye-lids	*o*		62
oligodactyly	*ol*	I	48
pink-eye series (type, Japanese ruby, pink-eye)	$+p^r p$	I	65, 84, 85
pallid (pink-eye-2)	*pa*	V	77
pirouette	*pi*	III	88
polydactyly	*py*		50
rodless retina	*r*	IV	53
rex	*Re*	VII	16, 35
ruby	*ru*	XII	29, 38
piebald	*s*	III	1, 25, 65.

Recognized and Named Mutations—*Cont'd.*

			80
screw tail	*sc*		59, 71
short-Danforth	*Sd*		30, 32
short-ear	*se*	II	70
shaker-1	*sh-1*	I	64
shaker-2	*sh-2*	VII	24, 81
silver	*si*	IV	47
splotch	*Sp*		78
shaker-short	*st*		27
syndactyly (with shaker)	*sy*		49
brachyury series (type, brachyury, lethal, lethal alleles)	$+T\ t^{p}t^{1}t^{2}t^{3}$	IX	10, 23, 28, 31
head spotting	*te*		37
umbrous	*U*		2, 72
urogenital abnormalities	*ur*		33
undulated	*un*	V	91
waltzing	*v*	X	39
varitint waddler	*Va*		12
dominant spotting series (type, dominant spotting, viable dominant spotting)	$+W\ W^{v}$	III	19, 22, 43, 56, 68
waved-1	*wa-1*	XI	15
waved-2	*wa-2*	VII	35, 57, 81
wellhaarig	*we*	V	49
white	*Wh*	XI	4, 7

New Undescribed Mutations

brachypody	*bp*	60
ducky	*du*	83
gray†	*g*	90
mahogany†	*ma*	69
wobbly	*wo*	40

*Complete data are not available for all the mutants listed herein. In some cases simple segregation has not been proved. For some, complete data indicating a separate mutation from phenotypically similar mutations are not available. Some mutants e.g. pigtail, posterior reduplication are not included because the genetics is not completely known.

†Mutants lost.

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Key to Abbreviations

*	= see list of inbred strains for detailed description.
Charac	= characteristics; e.g., tumor incidence or characteristic abnormalities.
Fert	= fertility; three grades are indicated, high, medium, low.
Genet	= genetic constitution; only mutant genes are listed.
Inbr	= inbreeding; number of brother × sister generations; if written "7 + 12", this signifies that the strain was inbred for 7 generations at one laboratory, then for 12 generations at a second laboratory.
Maintained by:	under this heading the laboratory upon whose sub-strain the description is based is usually listed first, although in some cases description of this strain is based upon characteristics submitted by several laboratories.
Orig	= origin; person or laboratory originating or other details as to origin.
Refs	= references; name of journal, volume, and first page are given.
Subl	= sublines; important sublines showing known differences are listed. In some cases sublines listed as such by an investigator are not genetic sublines.
Symbol	= symbol or name used to designate the strain.
Syn	= synonyms for the name or symbol usually employed to designate the strain.
Ad Inf	= Additional information pertaining to the strain.

LIST OF INBRED STRAINS

(Name or symbol designating strain in bold face type; see "Key to Abbreviations")

83 Inbr: 65+. Fert: medium. Genet: *aabb*. Orig: Lathrop, inbred by MacDowell. Charac: abnormal eyes, 20% overlaps. Refs: *J. Exp. Zoo.* 41:45. Maintained by: Mac Dowell, Little & Hummel.

89 Inbr: 45. Fert: low. Genet: *bb*. Orig: Lathrop, inbred by MacDowell. Charac: Circlers, some overlaps. Refs: *J. Gen. Physiol.* 11:57. Maintained by: MacDowell.

129 Inbr: 15. Fert: high. Genet: A^wA^w ppc^bc^b. Orig: Dunn. Refs: *Proc. Nat. Acad. Sc.* 31:267. Maintained by: Runner.

101BAg Inbr: 49. Fert: high. Genet: +. Orig: Dunn. Refs: *J. Genet.* 33:443. Maintained by: Dunn & Schoenheimer.

1194 Inbr: 53. Fert: medium. Genet: +. Orig: Lathrop. Charac: 4.6% lung tumor, 7% mammary tumor, a few tumors in other organs. Refs: *J. Exp. Med.* 43:339. Maintained by: Lynch.

A Inbr: 70+. Fert: medium. Genet: *aabbcc*. Orig: Strong, from cross made in 1921 of albino from Cold Spring Harbor and Bagg Albino; majority of sublines derived from Bittner's stock obtained from Strong in 1927. Charac: Breast tumors, high in breeders, low in virgins; lung tumors, high in breeders and virgins. Produces 5 to 10% young with cleft palate. Renal disease amyloidosis 100% at 15 to 18 months. Refs: *Am. J. Canc.* 15:2202, *Canc. Res.* 1:113, *J. Hered.* 27:21, *J. Nat. Cancer Inst.* 5:17, *A.A.A.S. Publ.* 22. Maintained by: Bittner, Heston, Rusch, Bacharach, Fraser, Gorer, Shabad, Warner, Dougherty. Imp. Cancer Res. Fund, W. S. Murray, Bonser, Dmochowski, Woolley.

Ak Subl: Aka, Akb, Akm, etc. Inbr: since 1930. Genet: albino. Orig: J. Furth and associates. Charac: leukemia stock (60-80%); other tumors rare; the leukemias are lymphoid or myeloid. Refs: *Am. J. Canc.* 19:521, *Canc. Res.* 1:99. Maintained by: Furth, Burchenal, Kemp, Shabad, Carr, Gross, Skipper.

Ax Inbr: 31 (since fostering). Fert: medium. Genet: *cc*. Orig: Bittner. From strain A (1934) fostered by CBA. Charac: low mammary tumor inc.; high lung tumor inc. Refs: *Science* 84:162. Maintained by: Bittner.

Anoph B Inbr: 22. Fert: high. Genet: *bbddey-1 ey-1 ey-2 ey-2*. Orig: Little. Charac: 90% completely anophthalmic. Refs: *J. Morph.* 68:279, *Genetics* 27:309, *J. Comp. Neurology* 83:121. Maintained by: Chase.

Ba Inbr: ?+59. Fert: low. Genet: albino. Orig: from original Bagg stock but differentiated at Iowa State for typhoid susceptibility. Maintained by: Gowen.

Bagg Inbr: 46+. Genet: albino. Orig: original "Bagg albinos," obtained from animal dealer in Ohio in 1913. Charac: some polydactylism; about 5% breast tumor in breeding females. Refs: *Am. Nat.* 50:222, *Arch. Psychology* No. 43, *Am. Nat.* June 1926. Maintained by: W. Murray, Warner.

Bagg L Inbr: 36. Fert: medium. Genet: *aabbcc*. Orig: from Bagg stock, via Strong, but maintained at the Rockefeller Institute as a distinct strain since 1921. Some of the original animals obtained from Strong carried agouti. Charac: 26% lung tumor (in mice of all ages), 4% mammary tumor in bred females, a low incidence of tumors in other organs. Refs: *J. Exp. Med.* 43:339, *Occas. Pub. A.A.A.S.* No. 4:22. Maintained by: Lynch.

B alb Bagg albino. Inbr: 73. Fert: high. Genet: *bbcc*. Orig: Bagg stock, inbred by MacDowell. Charac: tame, good foster mothers. Refs: *P.S.E.B.M.* 20:93, *J. Gen. Physiol.* 11:57, *Ibid.* 13:529. Maintained by: MacDowell.

B alb C Syn: C. Inbr: 56. Fert: medium. Genet: *bbcc*. Orig: Jackson Lab. subline of B alb strain. Charac: leukemia inc. high in some sublines. Lung tumor 20-30%, low breast tumors, moderately aggressive. poor fighting ability. Refs: *P.S.E.B.M.* 20:93, *Pub. Health Rep.* 53: 1647, *J. Nat. Canc. Inst.* 1:147. Maintained by: Snell, Andervont, Green, Heston, W. L. Russell, Runner, Scott, Rusch, Bittner, Gates, Law.

BTwb Inbr: 30. Fert: high. Genet: a^ta^t. Orig: Dunn, Charac: black-and-tan with white belly; rather weak. Refs: *Proc. Nat. Acad. Sci.* 14:816. Maintained by: Dunn and Schoenheimer.

C (see B alb C).

C3H Inbr: 95+. Fert: medium. Genet: +. Orig: Strong, from cross of dba and Bagg albino; extensive subline maintained by Bittner. Charac: breast tumor incidence high in breeding and virgin ♀♀; Andervont reports also high liver tumor incidences; lung tumor 5%. Refs: *Genet.* 20:586, *Am. J. Canc.* 35:614, *Canc. Res.* 1:115, *J. Nat. Canc. Inst.* 1:147, *A.A.A.S. Pub.* 22. Maintained by: Bittner, Andervont, Heston, Carr, Dougherty, Shabad, Tannenbaum, Pybus, Miller, Mider, Gross. Imp. Canc. Res. Fund, Goodale, Fekete, Runner, Scott, Vicari, Woolley, Dmochowski, Law, Jones.

C3Hb (Zb) Inbr: 20+ (since fostering). Fert: high. Genet: +. Orig: from C3H fostered on C57 black. Various lines at National Cancer Institute and Univ. of Minnesota, derived from separate foster nursed litters. Charac: low mammary tumors. Refs: *Cancer Res.* 3:441, 1:955, *A.A.A.S. Pub.* 22. Maintained by: Andervont, Bittner, Heston, Gross, Carr, Woolley.

C57 black Subl: 4, 6, 10, etc. Inbr: 35+. Fert: high. Genet: *aa*. Orig: Little, from heterozygous stock obtained from Miss Lathrop; much inbreeding prior to last 25 gens. b × s. Charac: breast tumors about 1% in bred ♀♀, 0 in virgins; various types of internal tumors, mostly sarcomas, 10-20%. Eye abnormalities in about 10% of animals, but incidence varying considerably in different sublines; hydrocephalus appears in some sublines. Low frequency of audiogenic seizures; males can be trained to be either pacific or aggressive; superior to C3H and B alb C in fighting. Refs: *J. Exp. Zool.* 41:45, *Am. J. Canc.* 36:431, *A.A.A.S. Pub.* 22. Maintained by: Russell, Andervont, Tannenbaum, Bacharach, Carr, Chase, Dougherty, Fraser, Gorer, Korteweg, Mider, Shabad, Warner, Woolley, Law, Fekete. Imp. Cancer Res. Fund, Bittner, Carworth Farms, Holman, Heston, Dmochowski.

C57 black A Orig: C57 black selected by Bagg for low mammary tumor. Charac: no mammary tumors for last 13 generations. Maintained by: Andervont.

C57 black T Inbr: 20+. Fert: high. Genet: a^ta. Orig: mutation in C57 black at Jackson Laboratory. Maintained by: Little and Hummel.

C57 brown Subl: *a* and *cd*. Inbr: 65+. Fert: medium. Genet: *aabb*. Orig: Little, derived from C57 black before this was homozygous for B. Charac: subline *a* like C57 black in tumor incidence; subline *cd* med. breast tumor incidence. Refs: *Am. Nat.* 67:278, *A.A.A.S. Pub.* 22. Maintained by: Heston, Vicari, Runner, Carworth Farms.

C57 leaden Inbr: 40+. Fert: medium. Genet: *aabblnln*. Orig: J. Murray, derived from C57 brown by mutation. Charac: tumor incidence like C57 black. 25% benign enlargement of mesenteric lymph node. Refs: *Am. Nat.* 67: 278, *A.A.A.S. Pub.* 22. Maintained by: Heston, Vicari, Dougherty, Gates.

C58 Inbr: 75+. Fert: medium. Genet: *aass*. Orig: Lathrop, inbred by MacDowell. Charac: tail and belly spots frequent; high leukemia. Refs: *Roux Archiv.* 109:549, *Am. J. Canc.* 26:85. Maintained by: MacDowell, Dougherty, Little and Hummell, Law, Snell.

CBA Inbr: 20+. Orig: Strong, via Jackson Lab. Maintained by: W. Murray, Bacharach, Carr, Gorer, Pybus, Miller, Andervont, Bonser, Kaplan.

cdE Inbr: 15. Orig: USDA. Genet: c^ec^e. Maintained by: Woolley.

ee Inbr: 35+. Fert: low. Genet: c^ec^e. Orig: Jackson Lab. Charac: low tumor, wide range of tumor types, high adrenal cortical carc. when ovariectomized at birth. Refs: *Canc. Res.* 5:193. Maintained by: Woolley, Vicari, Fekete, Law, Bittner.

CF1 Inbr: 20. Fert: medium. Genet: *aabbcc*. Orig: Carworth Farms. Charac: very susceptible to toxic action of sulfanilamides; resistant to typhoid (human or mouse); susceptible to *Streptococcus*. Maintained by: Carworth Farms.

CFCW Inbr: 15. Genet: *ccCaCa*. Orig: Mutant from CFW. Charac: Same as CFW. Maintained by Carworth Farms.

CFW Inbr: ?+21. Fert: high. Genet: *cc*. Orig: Rockefeller Institute (Webster). Charac: susceptible to typhoid and neurotropic viruses. Maintained by: Carworth Farms.

da. ab. Syn: Davenport ab. Inbr: 47. Fert: medium. Genet: *aabb*. Orig: out of C57. Charac: eyes abnormal to absent; skull and tooth abs. frequent. Refs: *Proc. Sixth Int. Genet. Cong.* 2:252. Maintained by: MacDowell, Little and Hummell.

dba Subl: 1 (subdivided into 111, 112, 122, etc.), 2 (subdivided into 212, 214, etc.), 4. Subl 1 and 2 separated 30 gens. ago following matings partly other than b × s; they differ in reaction to transplantable tumors. Subl 4 is closer to 2 than to 1, and behaves like 2. Inbr: 40+. Fert: high. Genet: *aabbdd*. Orig: Little. Charac: high breast tumor, leukemia 35-40% in ♀♀ of subline 212. Refs: *Am. J. Canc.* 20:573, *Canc. Res.* 1:564, *A.A.A.S.*

Pub. 22. Maintained by: Woolley, Andervont, MacDowell, Tannenbaum, Fekete, Vicari, Dougherty, Kemp, Korteweg, Mider, Warner, Heston, Carworth Farms.

E Inbr: 35. Fert: low. Genet: albino. Orig: Gowen. Charac: typhoid susceptible. Maintained by: Gowen.

Edinburgh Inbr: 32. Fert: low. Genet: *cc*. Orig: Dr. F. A. E. Crew, Univ. of Edinburgh in 1927. Charac: formerly high but now low incidence of liver hemangiomata and leukemia; occasional breast tumor. Refs: *Genetics* 17: 342, *J. Path. and Bact.* 54:155. Maintained by: Pybus and Miller.

F Inbr: 30+. Fert: low. Genet: +. Orig: from Strong via Kirschbaum 1945. Charac: approx. 55% leukemia at 400 days (lymphoid common, some myeloid). Refs: *Am. J. Cancer* 37:400. Maintained by: Kaplan, Kirschbaum.

Flex Inbr: 15. Fert: medium. Genet: *ccff*. Orig: Kamenoff. Charac: Flexed tail and anemia. Refs: *J. Morph.* 58:117. Maintained by: Dunn and Schoenheimer, Fraser.

G Inbr: 25. Fert: high. Genet: *aabbtitit*. Orig: unknown. Charac: cancer free. Maintained by: Kemp.

GFF Inbr: 16. Genet: fawn. Orig: Nat. Inst. Med. Res. via Agr. Res. Council Farm. Charac: large in size; very good for pharmacological and bacteriological work. Maintained by: Bacharach.

H Fert: high. Genet: albino. Orig: Howard, cross between Bagg albino and local white mice. Charac: well developed X zone in adrenal cortex. Refs: *Am. J. Anat.* 65:105, *Anat. Rec.* 77:181, *Endocrin.* 26:1048. Maintained by: Howard.

HD Inbr: 20+. Fert: low. Genet: *aabbHrhr*. Orig: Little, from dba × Dunn's hairless. Charac: medium tumor. Maintained by: Little and Hummel.

I Inbr: 50. Fert: medium. Genet: *ss*. Orig: Strong. Charac: spontaneous stomach lesion; spontaneous rectal prolapse. Refs: *Publ. Health Rep.* 53, 1947. Maintained by: Andervont, Dmochowski.

IF Inbr: 40. Fert: low. Genet: $a^t a^t$. Orig: Bonser. Selection of mice which developed papillomata early following application of carcinogens. Charac: Mammary tissue very sensitive to carcinogenic agents either distant or local. Refs: *J. Path. & Bact.* 46:581, 55:483. Maintained by: Bonser, Orr.

IFS Inbr: 25+. Fert: medium. Genet: $a^t a^t$. Orig: from Univ. of Leeds. Charac: low tumor incidence; a few lung tumors seen. Maintained by: Gorer.

IVB Inbr: 33. Fert: medium. Genet: *cc*. Orig: derived from Strain IV. Charac: medium breast cancer incidence. Ref: *C. R. Soc. Biol.* 104:1193. Maintained by: Dobrovolskaia-Zavadskaia.

JK Inbr: 50. Fert: low. Genet: *ppddbbsese*. Orig: from Strong. Charac: low mammary tumor incid. Refs: *J. Hered.* 28:40. Maintained by: Bittner.

Jerker Inbr: ?+10. Fert: low. Genet: *jejeAApp*. Orig: Grüneberg. Charac: headshaking, deafness, circling. Refs: *Proc. Nat. Acad. Sci.* 27:562. Maintained by: Dunn and Schoenheimer.

Kink Inbr: 10+8. Fert: high. Genet: *KiKicc*. Orig: Holman, via Dunn. Charac: kinky tail, waltzing, shaking, deafness. Refs: *J. Hered.* 31:427. Maintained by: Dunn and Schoenheimer.

KL Inbr: 15. Fert: medium. Genet: *cc*. Orig: cross of Swiss albino line × Aka line. Charac: high insulin tolerance. Maintained by: Chase.

L Inbr: 35. Fert: low. Genet: *aatiti*. Orig: Iowa State College and Gowen. Charac: differentiated for typhoid susceptibility. Refs: *J. Infect. Dis.* 78:79. Maintained by: Gowen.

L (fifty) Inbr: 15? Fert: high. Genet: *cc*. Orig: ♀ XIX × ♂ XLIII. Charac: high breast tumor incidence. Refs: *Acta du Int. Cancer* 5:55. Maintained by: Dobrovolskaia-Zavadskaia.

Large white Fert: high. Genet: albino. Orig: Mount Hope. Charac: ave. weight 60% greater than ordinary albinos. Refs: *J. Hered.* 29:101. Maintained by: Goodale.

Line 11 Inbr: 20+. Fert: high. Genet: $A^w A^w ss$. Orig: Dunn. Charac: small spot appeared by mutation and has been shown to be due to s. Maintained by: Dunn, Schoenheimer, Woolley, Snell.

Marsh Inbr: 74. Fert: high. Genet: albino. Orig: Marsh. Charac: mammary cancer. Refs: *Am. J. Canc.* 34:434. Maintained by: W. Murray, Warner, Andervont.

Misty Inbr: 30. Fert: high. Genet: *aabbddmm*. Orig: Woolley, mutation from dba 1, subline 12. Refs: *Am. Nat.* 75:507, *J. Hered.* 36:269. Maintained by: Woolley.

N Inbr: 25. Fert: low. Genet: *aabbddss*. Orig: Strong. Charac: low tumor incidence. Refs: *Quar. Rev. Biol.* 13: 51. Maintained by: Vicari.

NB Inbr: 26. Fert: low. Genet: $aabbc^{ch}p/c^{ch}pdse/dse$. Orig: Green, derived from cross of stocks from Gates and Margaret Hoover. Charac: bifurcated xiphisternum in 100% of individuals; leukemia frequent. Refs: *Genet.* 26: 192, *J. Hered.* 32:94. Maintained by: Green, Law, W. L. Russell.

NBT Inbr: 33. Fert: high. Genet: *cc*. Orig: subline of Simpson strain bred from mice with bone tumors. Charac: high incidence of bone tumors at first, now rare; susceptible to milk influence; one subline 20% leukemia; occasional stomach and skin papillomata. Refs: *Am. J. Canc.* 33: 98, 40:47. Maintained by: Pybus and Miller.

N-Bl Inbr: 11. Fert: high. Genet: *aabbddss*. Orig: Cloudman, cross between N and C57 black, line 6. Maintained by: Vicari.

NH Inbr: 20+. Fert: low. Genet: *ppddss*. Orig: from Strong via Kirschbaum. Charac: low mammary tumor incidence. Refs: *Cancer Res.* 2:531. Maintained by: Bittner.

O Inbr: 20. Fert: high. Genet: *cc*. Orig: from Dobrovalskaia-Zavadskaia. Charac: formerly high breast tumor incidence, now very low. Maintained by: Kemp.

O2O Leeuwenhoek-Huis Inbr: 60. Fert: medium. Genet: *ccaaBB*. Orig: Korteweg. Charac: low mammary cancer, pulmonary papillomas which eventually become metastasizing cancers. Refs: *Ned. Tydschr V. Gen.* 83:5989, *Am. J. Canc.* 38:506. Maintained by: Korteweg.

P Inbr: 36. Fert: medium. Genet: *aabbpprrdse/dse*. Orig: extracted following outcross of similar stock developed by Gates. Refs: *J. Hered.* 30:212, 32:94. Maintained by: Snell, Green, Dmochowski.

PL Inbr: +5. Fert: medium. Genet: *cc*. Orig: Rockefeller Inst. at Princeton, N. J. Charac: high leukemia incidence. Maintained by: Lynch and Murphy.

R111 Inbr: 20+. Fert: medium. Genet: *cc*. Orig: Dobrovolskaia-Zavadskaia. Charac: high breast tumor; Edinburgh subline: low mammary cancer; MH subline: low mammary, high lymphoma. Refs: *Compter Rendus* 101:518, *Bull. Canc.* 28:76. Maintained by: Woolley, Andervont, Law, Carr, Dobrovolskaia-Zavadskaia. Imp. Canc. Res. Fund, Bonser, Dmochowski.

RF Subl: Rfa, Rfb, etc. Inbr: since 1930. Fert: medium. Genet: albino. Orig: J. Furth and associates. Charac: low leukemia stock; lymphoid and myeloid 2%, monocytic leukemia 2%, latter is characteristic; lung tumors 32%; other neoplasms rare. Refs: *Am. J. Canc.* 19:521, *J. Exp. Med.* 69:1. Maintained by: Furth.

RI (Gowen) Inbr: ?+23. Fert: medium. Genet: albino. Orig: original stock Webster typhoid resistant; Iowa strain separated from original on basis of significantly greater resistance. Refs: *Genetics* 17:203. Maintained by: Gowen.

R.I.L. Syn: Rockefeller Inst. Leukemia. Inbr: 20+. Fert: medium. Genet: *aaBBcc*. Orig: Furth, via Rhodes via Lynch. Charac: (65% leukemia ♂♂; 80% leukemia ♀♀). Refs: *Cancer Res.* 4:622. Maintained by: Lynch, Law, Jackson Lab.

S Inbr: 39. Fert: low. Genet: albino. Orig: Iowa State College. Schott; derived from Schwing strain. Charac: high typhoid resistance. Refs: *J. Inf. Dis.* 71:131. Maintained by: Gowen.

Screwtail Inbr: 65 (15 since mutation). Fert: high. Genet: *Scsc*. Orig: B alb subline. Charac: scsc early mortality high, almost sterile. Refs: *J. Hered.* 33:435, *Anat. Rec.* 91:119. Maintained by MacDowell.

Se se-C Inbr: 16 (with forced heterozygosis for Se and se). Fert: high. Genet: $aabbc^{ch}c^{ch}DSe/dse$ and *dse/dse*. Orig: Green from cross of NB × B alb C. Charac: small mice. Refs: *Am. Nat.* 80:619. Maintained by: Green, Law.

Se se-Ab Inbr: 18 (with forced heterozygosis for Se and se). Fert: high. Genet: *bbDSe/dse* and *dse/dse*. Orig: Green from cross of P × B alb C. Charac: large mice. Refs: *Am. Nat.* 80:619. Maintained by: Green, W. L. Russell.

shaker-1 Inbr: 46. Fert: low. Genet: *bbcsh-1/csh-1*. Orig: from B alb. Maintained by: MacDowell.

Short-tailed Inbr: 11. Fert: medium. Genet: Tt^0 or t^1. Orig: Dunn. Charac: tailless genotype, short-tailed phenotype due to presence of modifiers. Maintained by: Dunn and Schoenheimer.

Simpson (S) Inbr: ?+24. Fert: medium. Genet: *cc*. Orig: Pybus and Miller subline of Simpson's strain 2. Obtained from Dr. Simpson, Buffalo, New York. Charac: low breast tumor incidence, occ. lung carc. and hepatoma. Refs: *Cancer Res.* 13:313, *Newcastle Med.* 514:151, *Am. J. Canc.* 33:98. Maintained by: Pybus and Miller, Imp. Cancer Res. Fund.

Snowy Inbr: 12. Fert: medium. Orig: wild mouse caught in Va. crossed with A^w and A stocks. Charac: high

agouti back with sharp flank line; feet white. Refs: *J. Hered.* 37:31. Maintained by: Woolley.

Splotch Inbr: 12+20. Fert: high. Genet: *aaSpsp*. Orig: W. L. Russell, from C57 black. Charac: heterozygotes white spotted; homozygotes die at 13 to 14 days and have kinky tail, spina bifida and other anomalies. Maintained by: Dunn and Schoenheimer.

Sto-Li Inbr: +70. Fert: medium. Genet: *aabbddPP*. Orig: MacDowell, Little's dba × Dunn piebald. Charac: low tumor incidence. Refs: *J. Canc. Res.* 14:434. Maintained by: MacDowell.

Street Inbr: 20+. Fert: high. Genet: *cc*. Orig: Engelbreth-Holm. Charac: some adenocarcinoma of glandular stomach. Maintained by: Engelbreth-Holm, Heston.

Swiss Subl: several sublines. Inbr: 35+. Fert: medium. Genet: albino. Orig: A. de Coulon, Lausanne, Switzerland; all "Swiss" mice are descended from this stock, though any separated prior to inbreeding may be rather different genetically from Lynch strain. Charac: 44% lung tumor in mice of all ages (80% in mice over 18 mos.), 19% mammary tumor in breed. ♀♀; few tumors in other organs. Refs: *P.S.E.B.M.* 43:186. Maintained by: Lynch, Woolley.

Waltzing Inbr: 100+. Fert: medium. Genet: *ssvv*. Orig: Gates Maintained by: Gates.

Waved-2 Inbr: ?+12. Fert: medium. Genet: *aasisiwa-2 wa-2*. Orig: obtained from Reed in 1941. Charac: medium susc. to induced pulmonary tumors. Maintained by: Heston.

White Label Inbr: 29. Fert: high. Genet: *cc*. Orig: obtained from Dr. Kreyberg, Oslo, 1934. Charac: mammary cancer in 20% breeding ♀♀. Refs: *J. Path. & Bact.* 52:263, 48:263. Maintained by: Bonser.

WR Inbr: 25. Fert: high. Genet: *ccAA*. Orig: from Webster; selected for resistance to *Sal. enteritidis*; inbred at Guy's Hospital. Charac: some imperforate vaginas; animals tend to obesity; susceptible to *B. piliformis*. Refs: *Am. J. Path.* 9:938, *J. Exp. Med.* 65:261. Maintained by: Gorer.

XVII Inbr: 13. Fert: high. Genet: *cc*. Orig: *Institut du* Radium. Charac: no tumor since 1937. Refs: *C. R. Soc. Biol.* 126:287. Maintained by: Dobrovalskaia-Zavadskaia.

XXXV Inbr: 22. Fert: medium. Genet: pc^e/pc^e. Orig: from U. S. through Miss Hardy. Charac: medium breast tumor incidence. Maintained by: Dobrovalskaia-Zavadskaia.

XXXIX Inbr: 28. Fert: high. Genet: +. Orig: Strong's CBA; high breast tumor incidence since 1945. Refs: *Bull. Cancer* 27:308. Maintained by: Dobrovalskaia-Zavadskaia.

XLII Inbr: 29. Fert: low. Genet: *lnlnaa*. Orig: J. M. Murray. Charac: no tumors since 1932. Refs: *Bull. Cancer* 27:308. Maintained by: Dobrovalskaia-Zavadskaia.

XLV Inbr: 26. Fert: medium. Genet: *cc*. Orig: ♀ RIII × ♂ XXXVIII. Charac: medium breast tumor incidence. Refs: *Acta Un Inst. Cancer* 5:55. Maintained by: Dobrovalskaia-Zavadskaia.

XLVIII Inbr: 14. Fert: high. Genet: *cc*. Orig: ♀ RIII × ♂ XLIII tail-less. Charac: medium breast tumor incidence; rare brachyury. Maintained by: Dobrovalskaia-Zavadskaia.

XLIX Inbr: 15. Fert: high. Genet: *dd*. Orig: ♀ RIII × ♂ XXXIII tail-less. Charac: high breast tumor incidence; rare brachyury. Maintained by: Dobrovalskaia-Zavadskaia.

Y Inbr: 30+. Fert: medium. Genet: $A^y a$. Orig: Little. Charac: some sarcomas; lung tumor 10-20%; amyloidosis and renal disease reported. Refs: *J. Exp. Med.* 59:229, *A.A.A.S. Publ.* 22. Maintained by: Woolley, Andervont, Heston, Law, Runner, Holman, Dmochowski.

Z Inbr: 35. Fert: high. Genet: albino. Orig: Gowen. Charac: differentiated from Swiss for high fertility; moderate typhoid resistance. Maintained by: Gowen.

Zrd Inbr: 16. Fert: medium. Genet: *aabbddpprsey-1 ey-1 ey-2 ey-2*. Orig: cross of anophthalmic line with other mutants to obtain line of recessives. Charac: very dilute brown, spotted and eyeless. Maintained by: Chase.

LOCATION OF WORKERS AND STOCKS

(Workers and name or symbol of stock in bold face type; see "Key to Abbreviations")

H. B. ANDERVONT, National Cancer Institute, Bethesda, Maryland
dba*, C57 black*, C3H*, C3Hb*, Y*, RIII*, B alb C*, I*, Marsh*, CBA*

J. J. BITTNER, Department of Physioldgy, Medical School, University of Minnesota, Minneapolis, Minn.
A*, Ax*, C3H(Zx)*, C3H(Zb)*, C57 black* (4, 6, B & D), dba* (12, 212, 7 & 8), ee*, JK*, NH*, I*, B alb C*

GEORGINA M. BONSER, School of Medicine, University of Leeds, England
A*, RIII*, CBA*, white label*, IF*

J. H. BURCHENAL, Sloan-Kettering Inst. for Cancer Research, New York, N. Y.
Akm*

J. G. CARR, Cancer Section, Institute of Animal Genetics, West Mains Road, Edinburgh
C57 black*, CBA*, C3H*, C3Hb*, RIII (Edinburgh & London sublines)*, A*, Akl*
YCBA Genet: *CB* (A^y or *A*). Inbr: 10. Ad Inf: A^y backcrossed to CBA each generation.
YRIII Genet: *B* (A^y or *A* and *C* or *c*). Inbr: 10. Ad Inf: A^y backcrossed to RIII each generation.
CY57 Genet: $A^y a$ or *aa*. Inbr: 6. Ad Inf: A^y backcrossed to C57 each generation.

CARWORTH FARMS, INC., New City, Rockland County, New York
CF_1*, CFW*, CFWC*, C57 black*, C57 brown*, dba (1)*

H. B. CHASE, Department of Zoology, Brown University, Providence, R. I.
Anoph B*, KL*, C57 black (6 & 10)*, Zrd*

C. H. DANFORTH, Department of Anatomy, Stanford University, California
Ruby eyes Genet: *ruru*. Inbr: 25 (close relative). Orig: Danforth. Ad Inf: eyes red not brown or pink; tumors few. Refs: *Proc. Nat. Acad. Sci.* 31:343.
Heterochromia iridis Genet: *ruru*. Inbr: 25 (close relative). Orig: Danforth. Ad Inf: eyes pink at birth; asymmetrically pigmented in adult.
Chinchilla Genet: $c^{ch}c^{ch}BB$. Inbr: 10 years. Ad Inf: no tumors ever observed.

L. DMOCHOWSKI, Dept. Expt. Pathology & Cancer Research, University of Leeds, England
Y*, P*, I*, C57 black*, A*, RIII, C_3H*

N. DOBROVALSKAIA-ZAVADSKAIA, Department of Genetics, Institute du Radium, 26 rue d' Ulm, Paris 5, France
XVII*, XXXV*, XXXIX*, XLII*, XLX*, XLVIII*, XLIX*, L*, IVB*, RIII*

ELLSWORTH C. DOUGHERTY, Donner Laboratory, Division of Medical Physics, Berkeley, California
C57 black (6)*, C58*, A*, dba (1 & 2)*, C3H*, C57 leaden*

L. C. DUNN and S. GLUECKSOHN-SCHOENHEIMER, Columbia University, New York, N. Y.
101 BAg*, BTwb*, Line II*, flex*
Brachy ex outcross Genet: T+. Inbr: 2. Orig: T gene (Dobrovalskaia-Zavadskaia) on spotted background. Ad Inf: heterozygotes short-tailed; homozygotes lethal. Refs: *C. R. Soc. Biol. Paris* 97:114.
fused subline on CF background Genet: *Fu+cc*. Inbr: 7. Orig: Reed. Ad Inf: fused tail. Refs: *Genetics* 22:1.
kink Genet: *Ki+cc*. Inbr: 9. Orig: Dunn, from Holman. Ad Inf: kinky tail, waltzing, deafness. Refs: *J. Hered.* 31:427.
jerker Genet: *jeje AA pp*. Inbr: ?+10. Orig: Grüneberg. Ad Inf: headshaking, deafness, circling. Refs: *Proc. Nat. Acad. Sci.* 27:562.
splotch Genet: *Spsp*. Inbr: ?+3. Orig: Russell. Refs: *Genet. Soc. Am.* 15:65.
Sd Genet: *sd+ cc*. Inbr: 2. Orig: Dunn, from Danforth. Ad Inf: heterozygotes short-tailed or tailless with abnormal kidneys; homozygotes lethal. Refs: *J. Hered.* 31:343.
Tailless 29 Genet: Tt^1. Inbr: 3. Orig: Dunn. Ad Inf: tailless, balanced lethal line. Refs: *Genetics* 24:587.
tailless A Genet: *Tt*. Inbr: 2. Orig: Dunn. Refs: *Genetics* 21:525.
Shorttailed Tt^n Genet: Tt^n. Inbr: 11. Orig: Dunn. Ad Inf: tailless genotype, short-tailed phenotype due to modifiers, (t^n = either t^0 or t^1).
subline "9059 Brachy" (abnormal urogenital system) Genet: *ur+ T+*. Inbr: 6. Orig: Dunn. Ad Inf: homozygotes lethal; extreme abnormalities in posterior trunk region. Refs: *J. Exp. Zool.* 104:70.
subline "9059" normal (abnormal urogenital system) Genet: *ur+ ++*. Inbr: 6. Orig: Dunn. Ad Inf: homozygotes lethal; abnormalities variable.
ruby (3 sublines) Genet: $ruruAa^t$ or *AA*. Inbr: 8. Orig: Dunn, from Danforth. Ad Inf: dilution of pigment; occasional heterochromia iridis. Refs: *Proc. Nat. Acad. Sci.* 31:343.

O. N. EATON, U. S. Department of Agriculture, Beltsville, Maryland
Snowy*

E. FEKETE, (See Jackson Lab.)

MIRIAM FINKEL, Argonne National Laboratory, Chicago, Illinois

C_3H*, A*

R. A. FISHER, Department of Genetics, University of Cambridge, England

Line 1. Genet: A^yA or a^t $\underline{W\ s}$ U fz (lx)

Line 2. Genet: $\underline{W\ s}$ Ca $\underline{Sd\ ln}$ (Va)

Line 3. Genet: $\underline{ru\ je}$ Re $wa\text{-}1$ bt (fz)

Line 4. Genet: Sd py b $\underline{se\ d}$ (fz)

Line 5. Genet: Aa^t or a $\underline{wa\text{-}1\ ln}$ b $sh\text{-}1$

Line 6. Genet: py $wa\text{-}1$ v U (Va)

Line 7. Genet: py s c^{ch} si Re

Line 8. Genet: cc^{ch} $wa\text{-}2$ $sh\text{-}2$ Fu $(fz\ Va)$

Line 9. Genet: $sh\text{-}2$ ln Re se U

Line 10. Genet: m b si $\underline{Ca\ bt}$ Fu (U)

Line 11. Genet: te p $\underline{Sd\ je}$ U

Line 12. Genet: $wa\text{-}2$ s $\underline{m\ b}$ v (je)

Line 13. Genet: si Sd $\underline{wa\text{-}1}$ $sh\text{-}2$ (lx)

Line 14. Genet: si $\underline{se\ d}$ Va Aa^t or a (je)

Line 15. Genet: si $\underline{ln\ v}$ $(te\ fz)$

Line 16. Genet: $\underline{Ca\ bt}$ c^ec^{ch} $\underline{se\ d}$ v (lx)

Line 17. Genet: $\underline{Fu\ se\ d}$ $wa\text{-}1$ te s

Line 18. Genet: py $\underline{pa\ wa\text{-}2}$ $sh\text{-}2$ te bt

Line 19. Genet: py ln $\underline{Fu\ ruje}$ (lx)

Line 20. Genet: Aa Re Fu Sd v

Line 21. Genet: Va lx $(\underline{Re\ m\ b}$ $te)$

These mice stocks are organized in a series of inbred lines, each segregating for a number of chosen factors. As fresh linkages are discovered these factors are continuously being modified and inbreeding only progresses far when the panel is unchanged for some time. Apart from the introduction of new factors each line is however, a closed system, normally propagated by sib matings.

F. C. FRASER, Department of Genetics, McGill University, Montreal, Canada

A*, C57 black*

rhino Genet: $hr^{rh}(Hr^{rh})$ $BbCca^ta^t$. Inbr: 5. Orig: Howard. Ad Inf: hair loss associated with follicular hyperkeratosis. Refs: *J. Hered.* 31:467.

hairless-piebald Genet: $HrhrSsbba^ta^t$. Inbr: 6. Orig: McGill.

naked Genet: $NnCcBba^ta^t$. Inbr: 5. Orig: McGill.

waved-2, shaker-2 Genet: $wa\text{-}2$ $sh\text{-}2/wa\text{-}2$ $sh\text{-}2$. Inbr: 3. Orig: McGill.

flex Genet: $ffcc$. Inbr: 2: Orig: Columbia, 1946.

J. FURTH, Southwestern University Medical School, Dallas, Texas

Ak*, Rf*

H. D. GOODALE, Mt. Hope Farm, Williamstown, Mass.

C3H*

head dot Genet: a^ta^tdd. Orig: Keeler.

large white

white tail Genet: aa. Orig: Mt. Hope.

Line 284 Genet: varied with much white. Orig: Mt. Hope. Ad Inf: by selection from head dot.

belly spot Genet: aa. Orig: Mt. Hope. Ad Inf: by selection from head dot.

W. H. GATES, Louisiana State University, Baton Rouge, Louisiana

B alb C*, C57 leaden*

dilution, short-ear Genet: $aabbddppsese$. Inbr: 20+. Orig: Gates.

hairless Genet: $aahrhr$. Inbr: 25+. Orig: Little.

Dom. Wt. Genet: $aaWw$. Inbr: 20+. Orig: Dunn.

piebald Genet: $aass$. Inbr: 20+. Orig: Danforth.

Dom. Wt.-piebald Genet: $aassWw$. Inbr: 20+. Orig: Danforth.

waltzing Genet: $aaddssvv$. Inbr: 20+. Orig: Little.

chinchilla Genet: $bbppc^{ch}c^{ch}$. Inbr: 20+. Orig: Little.

silver Genet: $aasisi$. Inbr: 20+. Orig: MacDowell. Ad Inf: carries dwarf.

waved-1 Genet: $aawa\text{-}1$ $wa\text{-}1$ $hrhr$. Inbr: 20+. Orig: MacDowell.

yellow Genet: A^ya. Inbr: 10. Orig: Danforth.

ext. dilution Genet: $A^yac^ec^e$. Inbr: 5. Orig: Danforth.

A^{sb} Genet: A^wA^wbb. Inbr: 15. Orig: Schwartz.

P. A. GORER, Department of Pathology, Guy's Hospital Medical School, London

A*, C57 black*, CBA*, JFS*, NBT*, W.R.*

J. W. GOWEN, Department of Genetics, Iowa State College, Ames, Iowa

R.I.*, S*, L(sil)*, Z*, E*, Ba*

E. L. GREEN, Department of Zoology & Entomology, The Ohio State University, Columbus, Ohio

B alb C*, P*, NB*, Se-se-C*, Se-se-Ab*

LUDWIG GROSS, Research Department, Veterans Administration Hospital, Bronx, New York

C3H*, C3Hb*, Ak*

H. GRUNEBERG, Biometry Department, University College, London

congenital hydrocephalus Genet: segregation for c, a, d, f, and ch. Orig: Grüneberg. Ad Inf: lethal at birth. Refs: *J. Genet.* 45:1.

hydrocephalus-3 Genet: segregates for b and $hy\text{-}3$. Orig: Grüneberg. Ad Inf: semi-lethal; survivors sterile. Refs: *J. Genet.* 45:22.

fidget Genet: cc, segregates for fi. Orig: Grüneberg. Ad Inf: viability reduced; ♀♀ useless. Refs: *J. Genet.* 45:22.

grey-lethal Genet: AA, segregates for gl. Orig: Grüneberg, from extreme dilute stock. Ad Inf: lethal at weaning. Refs: *Proc. Roy. Soc. B.* 118:321.

flexed-anemia Genet: $ddff$. Orig: Little. Refs: *J. Genet.* 43:45.

W^v Genet: aa segregates for b and W^v. Orig: Little & Cloudman. Ad Inf: W^vW^v usually sterile. Refs: *Proc. Nat. Acad. Sci.* 23:535.

undulated Genet: segregates for many genes. Orig: R. A. Fisher. An Inf: kyphosis of thoraic vertebrae; tail wavy.

shaker with syndactylism Genet: segregates for many genes. Orig: P. Hertwig. Refs: *ZIAV* 80:220.

anemia Genet: aa, bb. Segregates for d & an. Orig: P. Hertwig. Refs: *ZIAV* 80:220.

microphthalmia Genet: segregates for many genes. Orig: P. Hertwig. Ad Inf: not quite recessive; lethal at weaning; skeleton and teeth similar to gl but not allelic to gl. Refs: *ZIAV* 80:220.

W. E. HESTON, National Cancer Institute, Bethesda, Md.

A*, C3H*, dba(212)*, C57 black*, C57 brown*, C57 leaden*, B alb C*, Y*, Street*

waved-2 Genet: $aasiswa\text{-}2$ $wa\text{-}2$ Inbr: ?+12. Orig: Reed. Ad Inf: med. susc. to induced pulmonary tumors.

hairless Genet: $pphrhr$. Inbr: ?. Orig: Crew, through Carworth Farms.

WA Genet: $ffsh\text{-}2$ $wa\text{-}2/sh\text{-}2$ $wa\text{-}2$. Orig: Snell. Ad Inf: low fertility.

S. P. HOLMAN, Sarasota, Florida

C57 black*, Y*

KA Genet: cc. Inbr: +3. Orig: Kissel.

Hairless Genet: $hrhr$. Inbr: —. Orig: Kissel.

Naked Genet: Nn. Inbr: —. Orig: Jackson Lab.

Waved-1 Genet: $wa\text{-}1$ $wa\text{-}1$. Inbr: —. Orig: Keeler.

Shaker-1 Genet: $sh\text{-}1$ $sh\text{-}1$. Inbr: —. Orig: MacDowell.

Waltzing Genet: vv. Inbr: —. Orig: Gates.

Jerker Genet: $jeje$. Inbr: +3. Orig: Jackson Lab.

Twist Inbr: +10. Orig: Dunn.

Dom. spotting Genet: Ww. Orig: Dunn.

Silver Genet: $sisi$. Inbr: +3. Orig: Dunn.

Ruby eye Genet: $ruru$. Inbr: +3. Orig: Dunn.

Pink eye Genet: pp. Orig: Castle.

Het. Iridis Orig: Dunn.

Brachyury Genet: +T. Orig: Dunn.

Tailless Orig: Dunn.

Short-ear Genet: $sese$. Orig: Lathrop.

Chinchilla Genet: $c^{ch}c^{ch}$. Orig: Jackson Lab.

Black & tan Genet: a^ta^t. Orig: Ferrier.

Belted Genet: $btbt$. Inbr: +3. Orig: Jackson Lab.

Piebald Genet: ss. Orig: Castle.

EVELYN HOWARD, Johns Hopkins Medical School, Baltimore, Maryland

H*

IMPERIAL CANCER RESEARCH FUND, The Ridgeway, Mill Hill, London

A*, C57 black*, C3H*, RIII*, S (Simpson)*

R. B. JACKSON MEMORIAL LABORATORY, Bar Harbor, Maine—E. FEKETE

ce*, C3H*, C57 black (6)*, dba (1)*

R. B. JACKSON MEMORIAL LABORATORY, Bar Harbor, Maine—C. C. LITTLE and K. P. HUMMEL

85*, C58*, da. ab.*, HD*, C57 black T*

rodless Genet: rr. Orig: Dunn.

R. B. JACKSON MEMORIAL LABORATORY, Bar Harbor, Maine—M. RUNNER

129*, B alb C*, A*, C3H*, C57 black (6)*, C57 brown (cd)*, Y*

R. B. JACKSON MEMORIAL LABORATORY, Bar Harbor, Maine—E. S. RUSSELL

aaW^{v}w Genet: *aaW^{v}w*. Orig: Russell. Ad Inf: crosses of miscellaneous *aaW^{v}w* mice.
aaWw-C57bl Genet: *aaWw*. Orig: Russell. Ad Inf: *W* introduced into C57 black (4).
aaW^{v}w-C57bl Genet: *aaW^{v}w*. Orig: Russell. Ad Inf: *W^v* introduced into C57 black.
Agouti-C57bl Genet: *Aa*. Orig: Russell. Ad Inf: introducing *A* into C57 black.
A^{y}aW^{v}w Genet: *A^{y}aW^{v}w*. Orig: Russell. Ad Inf: introducing *A^y* and *W^v* genes into C57 black.
Maintaining color stocks carrying *A^{y}a* and *aa* in combination with *CC*, $c^{ch}c^{ch}$, $c^e c^e$, *BB*, *bb*, *DD*, *dd*, *PP*, *pp* in combinations from which all colors may be made up.

R. B. JACKSON MEMORIAL LABORATORY, Bar Harbor, Maine—j. P. SCOTT
B alb C*, C3H*, C57 black (10)*

R. B. JACKSON MEMORIAL LABORATORY, Bar Harbor, Maine—G. D. SNELL
B alb C*, CA*, C58*, P*, Line 11*
DY Genet: *Dydy*. Orig: Snell. Ad Inf: new recessive behavior mutation; low fertility and viability in homozygote.
RU Genet: *aajeru/jeru*. Orig: Snell.
M Genet: *btbt*. Orig: Murray. Ad Inf: medium fertility.
WH Genet: *Whwh*. Orig: Charles. Ad Inf: *Wh* being introduced into C57 black.
V Genet: *lnlnss wa-1 wa-1 Vv*. Orig: Snell.
WA Genet: *ffsh-2wa-2/sh-2wa-2*. Orig: Snell. Ad Inf: low fertility.
SD Genet: *Sdsd*. Orig: Dunn. Ad Inf: low fertility.
DW Genet: *Dwdw*. Orig: Snell.
Sil Genet: *aasisi*. Orig: MacDowell. Ad Inf: original English silver strain maintained by MacDowell for 60 generations.

R. B. JACKSON MEMORIAL LABORATORY, Bar Harbor, Maine—E. VICARI
A*, ce*, C3H*, C57 black (10)*, C57 brown (a)*, C57 leaden*, dba (2)*, N*, N-Bl*

R. B. JACKSON MEMORIAL LABORATORY, Bar Harbor, Maine—G. W. WOOLLEY
A*, ce*, C3H*, C3Hb*, C57 black (10)*, dba (2)*, Misty*, Swiss*, Y*, Line 11*, edE*
Pirouette Genet: *pipi*. Orig: Woolley. Ad Inf: mutation from C3H strain; linked with W on 3rd chrom.
Kreisler Genet: *Krkr*. Orig: Hertwig.
Anemia Genet: *Anan*. Orig: Hertwig. Ad Inf: anemic at birth, seldom live.
Fidget Genet: *Fifi*. Orig: Grüneberg. Ad Inf: ♀♀ fertile, ♂♂ sterile or poor breeders.
Fuzzy Genet: *fzfz*. Orig: Carnochan. Ad Inf: hair curled and fine texture.
Snowy Genet: *A^{w}A^w*. Orig: USDA. Ad Inf: light-bellied agouti with modifier producing white belly.
Snowy belly-2 Genet: *a^{t}a^t*. Orig: USDA. Ad Inf: producing light belly.

ELIZABETH E. JONES, Department of Zoology, Wellesley College, Wellesley, Massachusetts
C3H*

H. KAPLAN, National Cancer Institute, Bethesda, Maryland
F*, CBA*

TAGE KEMP, The University Institute for Human Genetics, Copenhagen
Aka*, O*, C*, dba*
Strain B Genet: segregates for many genes. Inbr: no. Orig: from Snell via MacDowell. Ad Inf: carries dwarfism.
Strain F Genet: *A^{y}a*. Inbr: no. Orig: from Cuenot.

R. KORTEWEG, Laboratorium Antoni Van Leeuwenhoek-huis, Amsterdam
C57 black*, dba*, 020 Leeuwenhoek-Huis*
P. Leeuwenhoek-Huis. Genet: *aa*. Inbr: 6+. Orig: Korteweg. Ad Inf: started from ♀ dba. 20 gen's. back-crosses to ♂♂ C57 black. b & s for 6 gens.

W. LANDAUER, Storrs Agricultural Experiment Station, Storrs, Connecticut
Brachypody Genet: *bpbpcc*. Orig: through Pappenheimer, Harvard.

L. W. LAW, National Cancer Institute, Bethesda, Maryland
ce*, C3H*, C3Hb*, C58*, dba*, RIL*, RIII*, NB*, Se-so-C*

C. C. LITTLE (See Jackson Lab.)

CLARA J. LYNCH, Rockefeller Institute Medical Research, New York
RIL*, P.L.*, 1194*, Bagg L*, Swiss*
E & EH Genet: *cc*. Inbr: pen inbred. Orig: Lathrop.
hairless Genet: *aabbpphrhr*. Inbr: pen inbred.
scant Genet: *aabbscsc*. Inbr: pen inbred. Orig: scant haired mutant in Bagg L stock.
flexed Genet: *sh-2 wa-2/sh2+ +f*. Orig: Hunt.

E. C. MacDOWELL, Cold Spring Harbor, New York
C58*, StoLi*, 89*, dba*, B alb*, Screw-tail*
Shaker-1 Genet: *sh-1 sh-1*. Inbr: 3. Orig: B alb subline after 48 generations crossed by B alb.

R. K. MEYER (See R. Speirs)

G. B. MIDER, School of Medicine, University of Rochester, New York
C57 black*, dba (2)*, C3H*

J. B. MURPHY, Rockefeller Institute Medical Research, New York, N. Y.
RIL*, P.L.*

W. S. MURRAY, Detroit Cancer Institute, Detroit, Mich.
A*, Bagg*, C57 black*, CBA*, dba*, Marsh*

C. M. OSBORN, Syracuse University, Syracuse, New York
Dwarf Genet: *Dwdw*. Orig: Snell.

F. C. PYBUS and E. W. MILLER, J. H. Burn Res. Lab., Royal Victoria Inf., Newcastle-on-Tyne, England
CBA*, C3H*, Edinburgh*, Simpson Strain 3*, NBT*

J. H. QUISENBERRY, Department of Genetics, Texas A & M College, College Station, Texas
Pink-eye (p)
intensifier Genet: *pp*. Inbr: 5. Orig: mutation from p. Ad Inf: darkens coat color in pink-eyes.

M. N. RUNNER (See Jackson Lab.)

H. P. RUSCH, McArdle Memorial Laboratory, University of Wisconsin, Madison, Wisconsin
A*, B alb C*

E. S. RUSSELL (See Jackson Lab.)

W. L. RUSSELL, Monsato Chemical Co., Clinton Laboratories, Oak Ridge, Tennessee
A*, C3H*, dba*, C57 black*, C57 brown*, C57 leaden*, B alb C*, Y*, NB*, Se se-ab*
Waved-2 Genet: *aasisiwa-2 wa-2*. Inbr: ?+12. Orig: Reed via Heston.
hairless Genet: *pphrhr*. Inbr: ?. Orig: Crew, through Carworth Farms via Heston.
WA Genet: *ffsh-2 wa-2/sh-2 wa-2*. Orig: Snell via Heston.

J P. SCOTT (See Jackson Lab.)

L. M. SHABAD, Laboratory of Oncology, Moscow, Baltusky Poselok, 13.
A*, C57 black*, Akb*
C3HA Genet: +. Inbr: 9. Orig: Moscow, from cross of ♀ C3H × ♂ A. Ad Inf: high incidence mammary tumors. Refs: *Bull. Expt. Biol. & Med.* 22:9.
CC57 Inbr: 7. Orig: Moscow, from cross ♀ B alb C × ♂ C57 black. Ad Inf: very sensitive to milk agent. Refs: *Arch. Biol.* 9:2, *Bull. Expt. Biol. & Med.* 22:29.

H. E. SKIPPER, Southern Research Institute, Birmingham, Alabama
Akb*

G. D. SNELL (See Jackson Lab.)

R. SPEIRS and R. K. MEYER, Department of Zoology, University of Wisconsin, Madison, Wisconsin
dba*, ce*, C57 black*, B alb C*, Y*

A. TANNENBAUM, Michael Reese Hospital, Chicago, Ill.
C57 black*, dba*, C3H*

E. VICARI (See Jackson Lab.)

S. G. WARNER, Biological Station, Springville, Erie County, New York
C57 black*, dba*, Marsh*, A*, B alb*

JOSEPH WARKANY, Children's Hospital, Research Foundation, Cincinnati, Ohio
Kinky tail Genet: *Kiki*. Inbr: ?+3. Orig: Dunn and Schoenheimer. Refs: *J. Hered.* 31:427.

G. W. WOOLLEY (See Jackson Lab.)

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